Device impiantabili ed apnee notturne: solo diagnosi o anche parte della terapia

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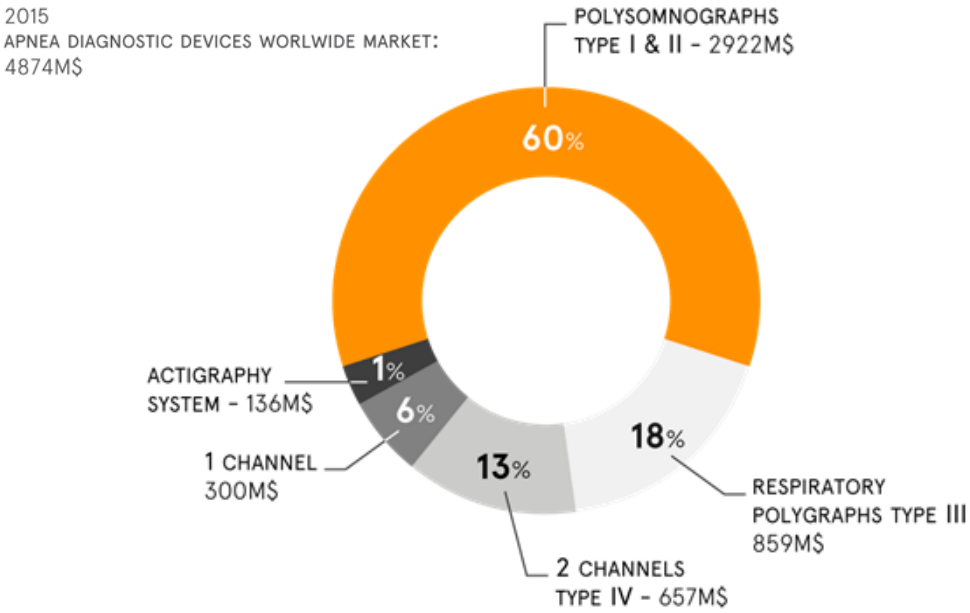
Diagnosis of sleep-disordered breathing



- PSG is the "Gold standard" for diagnosing sleep apnea
- Patients are referred to sleep apnea specialists and Pulmonologists for PSG
- Limited number of sleep laboratories available
- PSG may not be readily available as waiting lists are long and the cost is significant
- PSG does not allow us to identify the potential interaction between AF and SA

SAS is a prevalent, yet often undiagnosed pathology

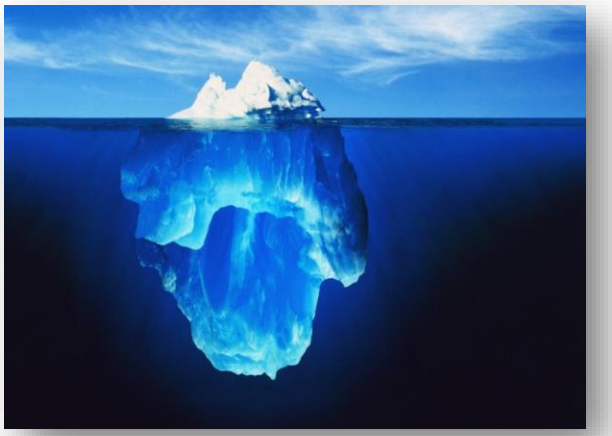
SLEEP *Fast Track Publication*

Estimation of the Clinically Diagnosed Proportion of Sleep Apnea Syndrome in Middle-aged Men and Women

Summary: The proportion of sleep apnea syndrome (SAS) in the general adult population that goes undiagnosed was estimated from a sample of 4,925 employed adults. Questionnaire data on doctor-diagnosed sleep apnea were followed up to ascertain the prevalence of diagnosed sleep apnea. In-laboratory polysomnography on a subset of 1,090 participants was used to estimate screen-detected sleep apnea. In this population, without obvious barriers to health care for sleep disorders, we estimate that 93% of women and 82% of men with moderate to severe SAS have not been clinically diagnosed. These findings provide a baseline for assessing health care resource needs for sleep apnea. **Key Words:** Sleep apnea—Epidemiology—Sleep disorders—Prevalence.

Sleep, 20(9):705–706

Between 50% and 90% of patients are often undiagnosed and untreated[1]



1. Lee W et al. Expert Rev Respir Med 2008

SLEEP

THE MEDICAL COST OF UNDIAGNOSED SLEEP APNEA

The Medical Cost of Undiagnosed Sleep Apnea

Kapur et al. Sleep 1999;22:749-755

Obstructive sleep apnea is an under-diagnosed, but common disorder with serious adverse consequences. Cost data from the year prior to the diagnosis of sleep-disordered breathing in a consecutive series of 238 cases were used to estimate the potential medical cost of undiagnosed sleep apnea and to determine the relationship between the severity of sleep-disordered breathing and the magnitude of medical costs. Among cases, mean annual medical cost prior to diagnosis was $2720 versus $1384 for age and gender matched controls ($p<0.01$). Regression analysis showed that the reciprocal of the apnea hypopnea index among cases was significantly related to log-transformed annual medical costs after adjusting for age, gender, and body mass index ($p<0.05$). We conclude that patients with undiagnosed sleep apnea had considerably higher medical costs than age and sex matched individuals and that the severity of sleep-disordered breathing was associated with the magnitude of medical costs.

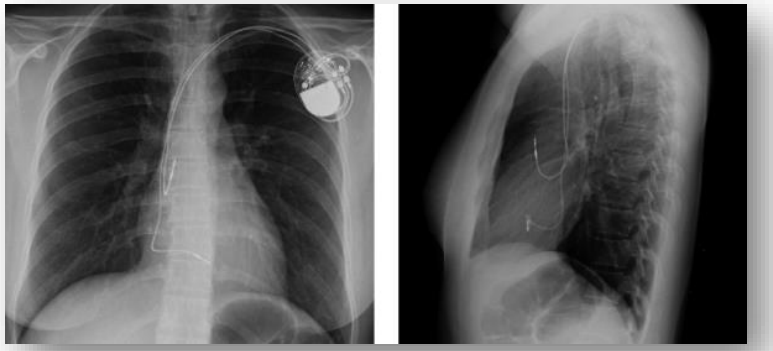
Using available data on the prevalence of undiagnosed moderate to severe sleep apnea in middle-aged adults, we estimate that untreated sleep apnea may cause $3.4 billion in additional medical costs in the U.S.

Whether medical cost savings occur with treatment of sleep apnea remains to be determined.

Jennum P, Kjellberg J. Thorax 2011.

High Prevalence of Sleep Apnea Syndrome in Patients With Long-Term Pacing

The European Multicenter Polysomnographic Study

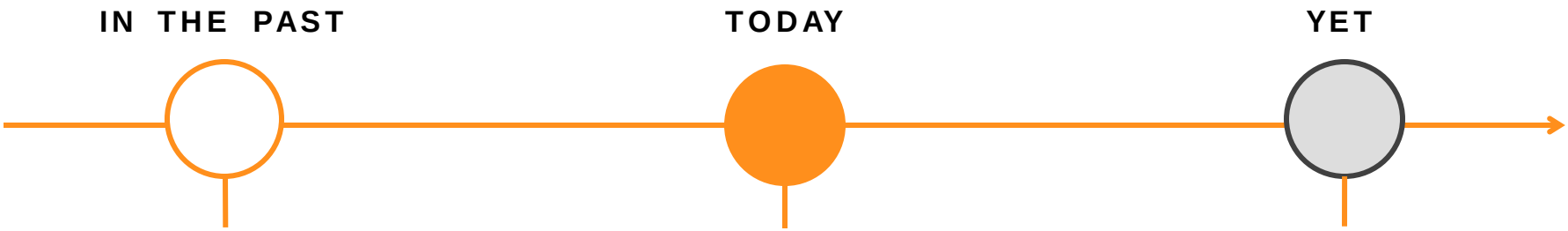


1. 60% dei pazienti portatori di pacemaker sono affetti da disordini del sonno.
2. Le forme ostruttive sono le più frequenti nei portatori di pacemaker.
3. I sintomi ed i disturbi tradizionalmente presenti nella popolazione OSAS, anche se severa, spesso non sono presenti nei pazienti portatori di pacemaker.
4. Non vi è chiara correlazione tra età, BMI e severità dell'OSAS nei portatori di pacemaker.

Uno screening sistematico in questi pazienti potrebbe aiutarci a slatentizzare i casi di OSAS severi e quindi a trattarli riducendo il rischio cradiovascolare.

Garrigue et al. Circulation. 2007;115:1703-1709

What have we learned so far?



OSA is prevalent in AF pts and among PM pts

-

There is a higher recurrence of AF if OSA remains untreated

-

Success of AF treatment depends on the severity of OSA

-

SAS therapy effective on symptoms, and on reduction of CV morbi-mortality

WHAT DO THE GUIDELINES & EXPERTS SAY?

> *Given recent data about the increased recurrence of AF in patients with untreated OSA and the lower risk of recurrence with CPAP therapy, it would be important to identify the large proportion of patients with AF who have OSA and are eligible for such treatment.* ***The presence of OSA should be considered in all patients with AF*** *and screening might be warranted in patients with AF who are obese or hypertensive.*[1]

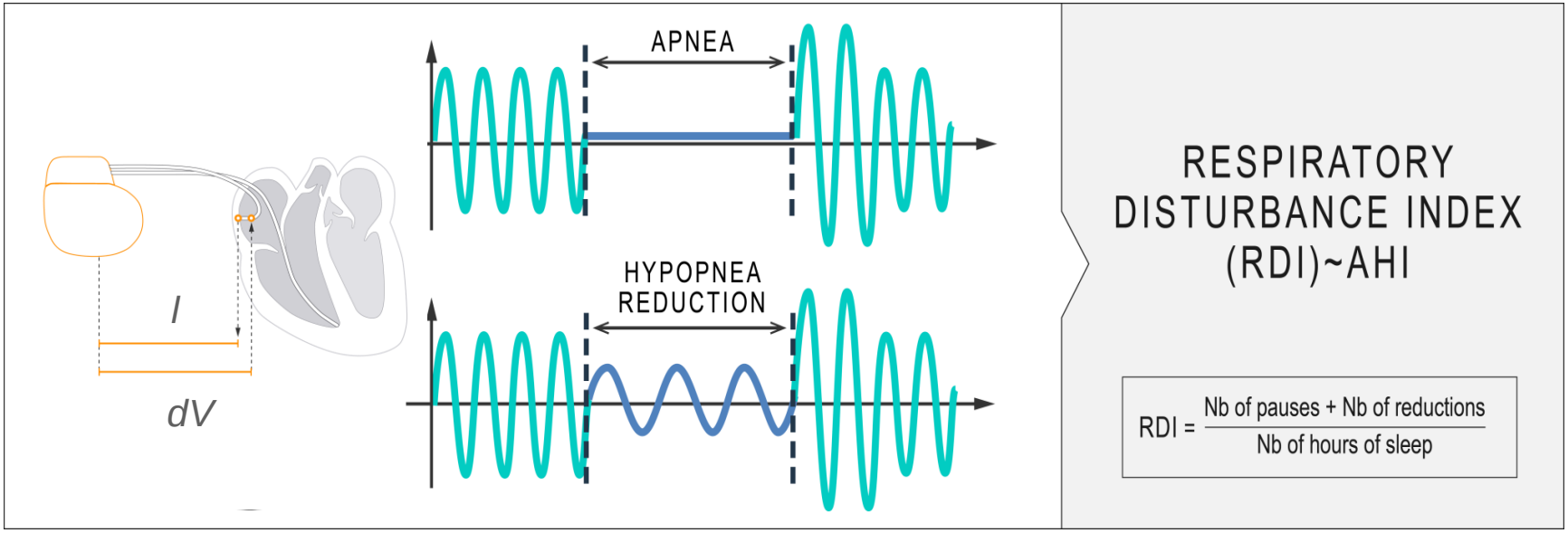
> It seems reasonable to **consider obstructive sleep apnea screening in AF patients** with risk factors. Obstructive sleep apnoea treatment should be optimized to improve AF treatment results in appropriate patients.[2]

1. Gami S A, Pressman G, Caples M S et al. Association of atrial fibrillation and obstructive sleep apnea. Circulation 2004 ; 110:364–367
2. Kirchhof P, Benussi S, Kotecha D et al. 2016 ESC guidelines for the management of atrial fibrillation. Eur Heart J 2016 ; 37: 2893-2962

SAM™ (Sleep Apnea Monitoring)

A UNIQUE SCREENING TOOL FOR SA AVAILABLE ON MCP CRM PM

- Sleep apnea is detected from the minute ventilation (MV) sensor that measures the transthoracic impedance [Zth (Ω) = dV (V) / I (A)]
- Every night SAM monitors the variation of trans-thoracic impedance to identify ventilation reductions or ventilation pauses
- Provides an indicator of the severity of SA, called RDI



A pacemaker transthoracic impedance sensor with an advanced algorithm to identify severe sleep apnea: The DREAM European study

Pascal Defaye, MD,[*] Ines de la Cruz, MD,[†] Julio Martí-Almor, MD, PhD,[‡]
Roger Villuendas, MD,[§] Paul Bru, MD,[‖] Jérémie Sénéchal, MSc,[¶]
Renaud Tamisier, MD, PhD,[#**] Jean-Louis Pépin, MD, PhD[#**]

(Heart Rhythm 2014;11:842–848)

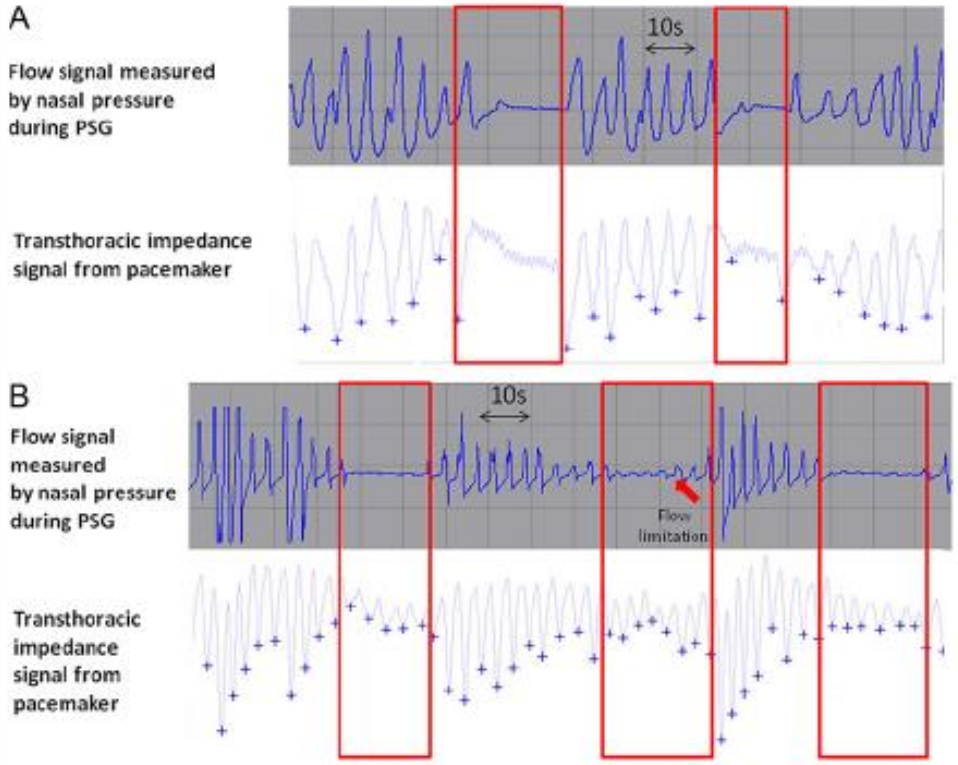


Figure 1 Nasal pressure measurements during polysomnography (PSG) and corresponding sleep apnea monitoring (SAM) recordings of transthoracic impedance from the minute ventilation sensor. **A:** Two typical apneas are framed. Apneas are identified clearly on the nasal pressure signal during PSG as a drop in the peak signal excursion by ≥90% of pre-event baseline. Note the concurrent reduction of the amplitude of the thoracic impedance signal from the pacemaker. **B:** Hypopnea events are framed. Hypopneas in adults are scored when the peak signal excursions drop by ≥30% of pre-event baseline using nasal pressure. Note the simultaneous reduction of the amplitude of the thoracic impedance signal from the pacemaker.

Obiettivo dello studio

The main objective of the study was to evaluate the performance of the SAM algorithm in comparison to PSG by using the following 2 approaches:

- An event-based approach, which assessed the performance of the SAM algorithm toward the detection of abnormal respiratory events, as determined from investigators' interpretation of PSG
- An index-based approach, which assessed the performance of the SAM algorithm toward the diagnosis of SA severity, as determined from the apnea-hypopnea index derived from PSG scoring

Risultati:

Event-based approach sensitivity of 60.4% and PPV of 50.6%

Index-based approach sensitivity of 88.9% and specificity of 84.6%

Main findings

In our unselected population of patients with PMs, we found a prevalence of 78% in moderate to severe SA and 56% in severe SA.

A pacemaker transthoracic impedance sensor with an advanced algorithm to identify severe sleep apnea: The DREAM European study

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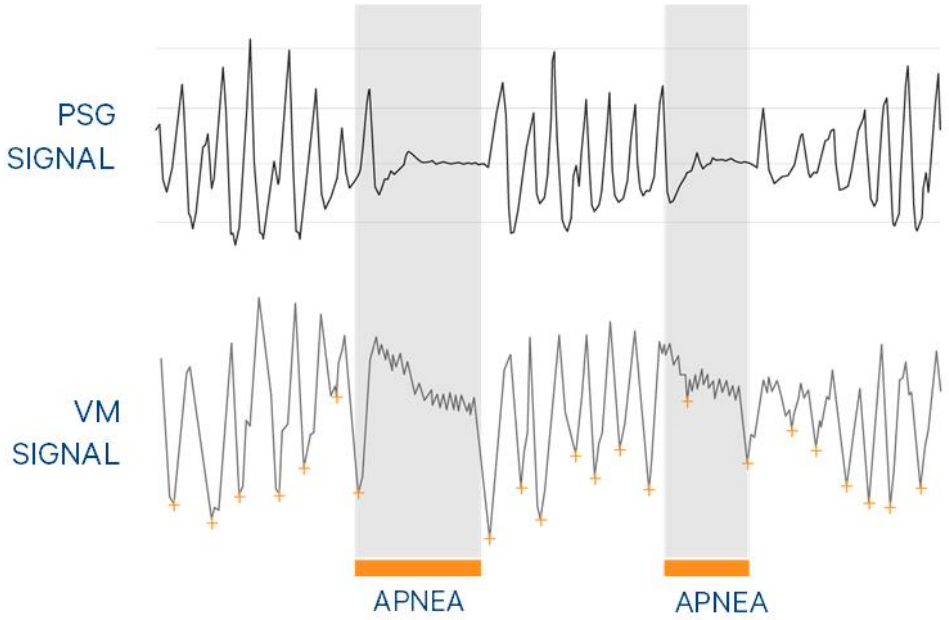
(Heart Rhythm 2014;11:842–848)

The **DREAM study** compared the AHI from PSG to the RDI from the pacemaker. Both AHI and RDI were acquired during the same night.

- A significant correlation between AHI-PSG and RDI-SAM was found in all patients ($r=0.81$, $p<0.001$).
- The optimal SAM-RDI cut-off value for detecting severe SA (PSG-AHI 30 events/h) is 20 events/h.
- SAM demonstrated strong performance with high specificity, PPV and NPV for severe sleep apnea compared to PSG.



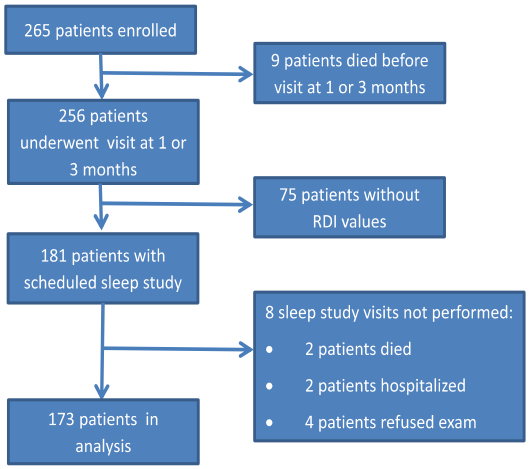
PERFORMANCE OF THE SAM ALGORITHM

SENSITIVITY	SPECIFICITY	PPV	NPV
88.9%	84.6%	88.9%	84.6%

Pascal Defaye & al., a pacemaker transthoracic impedance sensor with an advanced algorithm to identify severe sleep apnea: The DREAM European study Heart Rhythm 2014;11:842–848

Implantable cardioverter-defibrillator–computed respiratory disturbance index accurately identifies severe sleep apnea: The DASAP-HF study

Antonio D'Onofrio, MD,* Maria Teresa La Rovere, MD,† Michele Emdin, MD, PhD,‡§ Alessandro Capucci, MD, PhD,‖ Gianfranco Sinagra, MD, PhD,¶ Valter Bianchi, MD,* Ennio C.L. Pisanò, MD,# Paolo Pieragnoli, MD,** Maurizio Tespili, MD,†† Mario Luzi, MD,‖ Antonello Talarico, MD,‡‡ Massimo Zecchin, MD,¶ Antonio Rapacciuolo, MD, PhD,§§ Marcello Piacenti, MD,§ Ciro Indolfi, MD, PhD,‖‖ Miguel Angel Arias, MD,¶¶ Igor Diemberger, MD,## Catia Checchinato, MD,*** Giuseppe Boriani, MD, PhD,††† Luigi Padeletti, MD, PhD‡‡‡



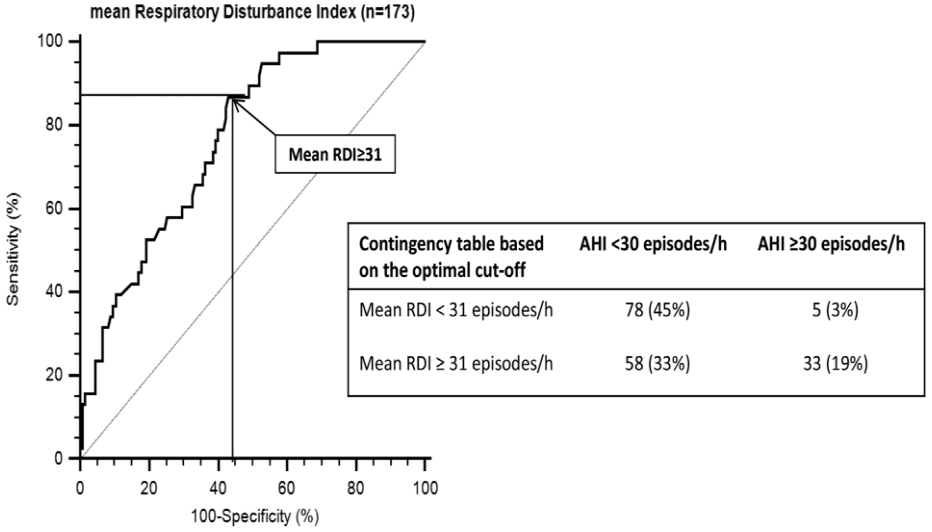
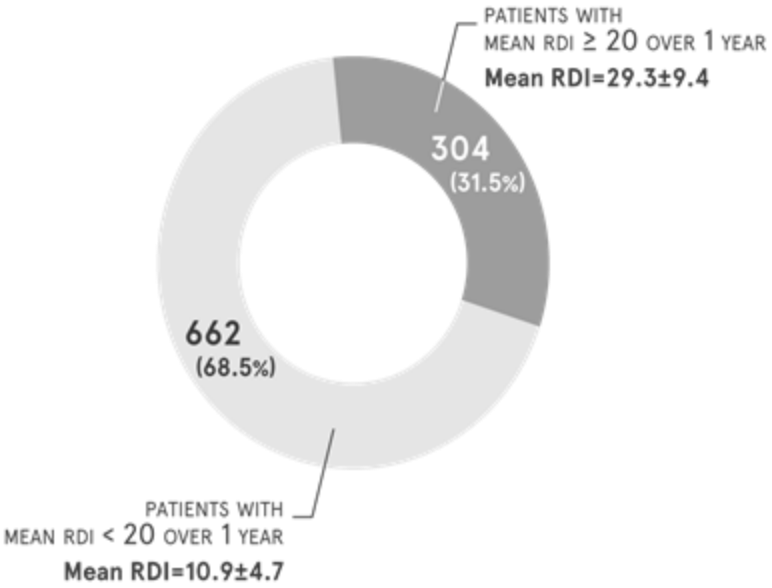


Figure 2 Receiver operating characteristic curve analysis of the RDI. Sensitivity and specificity were 87% (95% confidence interval 72%–96%) and 56% (95% confidence interval 48%–66%), respectively, at 31 episodes/h. AHI = apnea-hypopnea index; RDI = respiratory disturbance index.

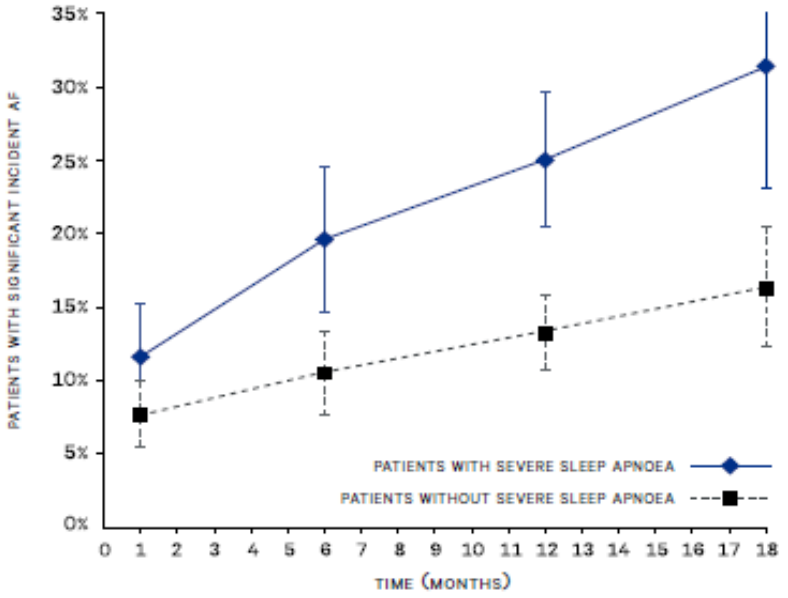
Results from the RESPIRE Study

Incidence of severe SA in unselected pacemaker population (N=966) of RESPIRE study over 1 year.



31.5% had severe sleep apnea with a mean RDI ≥ 20 over 1 year

Incidence of significant AF according to SA severity



~2x higher incidence of significant AF in severe sleep apnea PM patients

Europace 2017; 19(Suppl3): iii216

Results from the RESPIRE Study

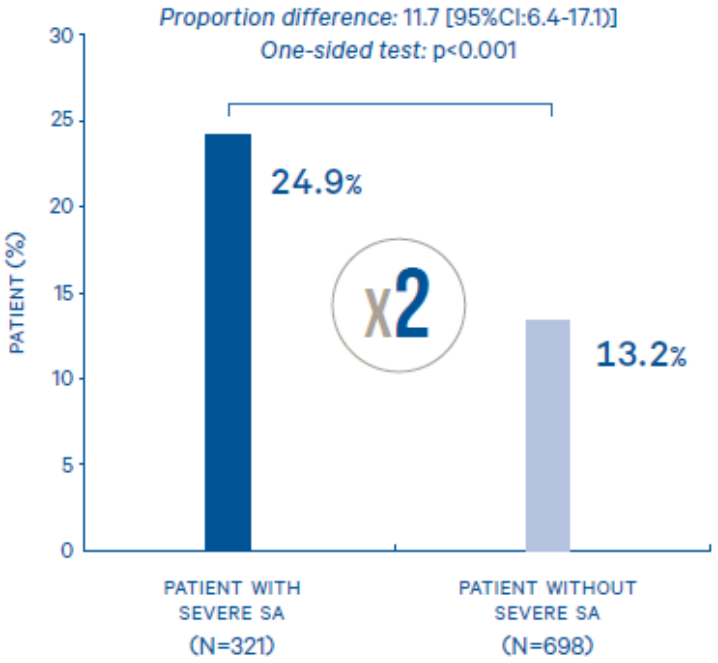
INCIDENCE OF SA AND ASSOCIATION WITH AF IN AN UNSELECTED PACEMAKER POPULATION WITH [SAM]™ ALGORITHM

Sleep Apnea burden

Severe Sleep Apnea burden (RDI>20) was identified in **31.1% of patients of an unselected pacemaker population**

AF risk

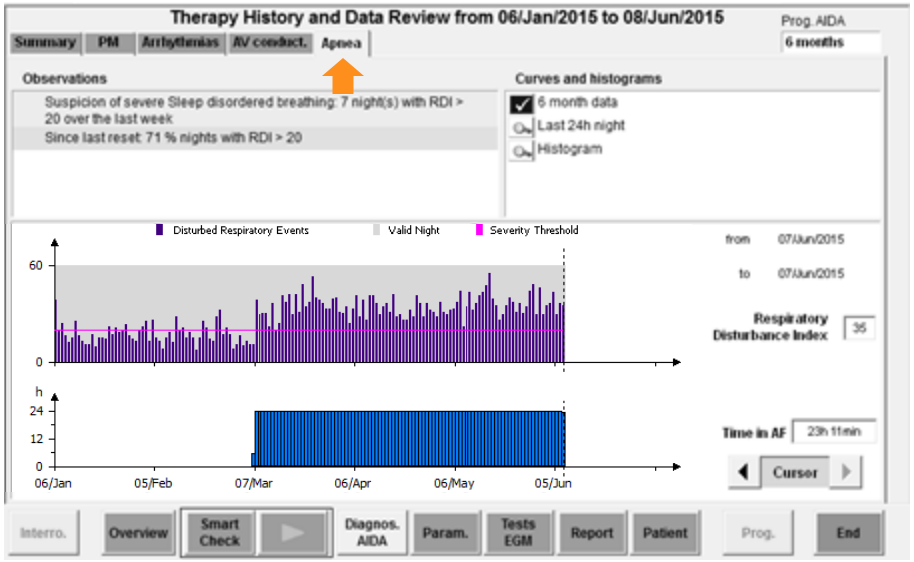
~2x higher incidence of significant AF in severe sleep apnea PM patients



Marti-Almor J et al.. Heart Rhythm. 2019 Sep 4.

SAM: easy to use tool

- Activated automatically
- Alert messages on the overview screen of the programmer
- Monitoring Period can be programmed

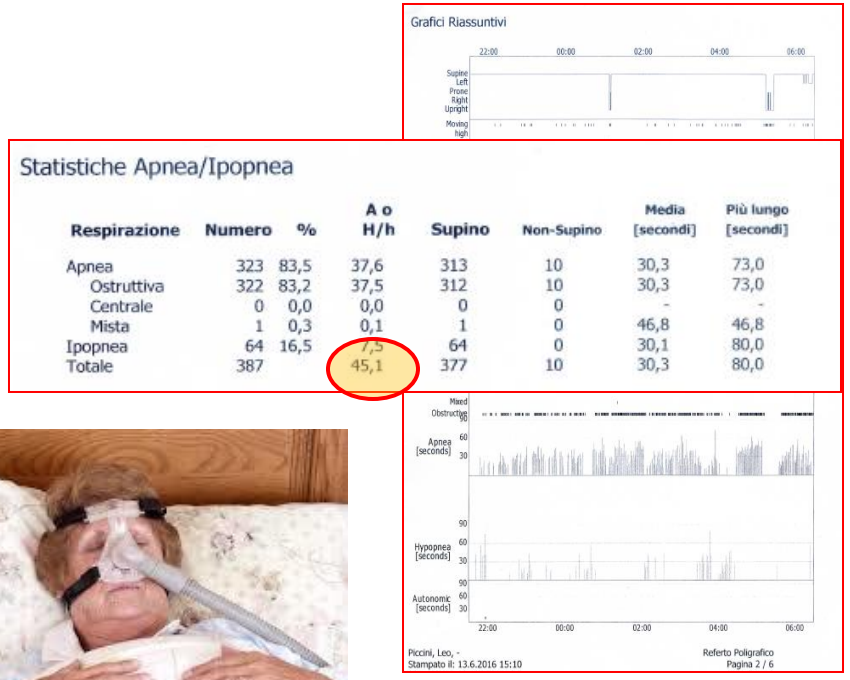


- Long period trend of RDI during follow up, with repeated measurements every single night
- A severity threshold (20 events/h)* indicates the risk of severe SAS
- Simultaneous display of AF and SA burdens

*Pascal Defaye & al., The DREAM European study Heart Rhythm 2014;11:842–848

Caso clinico

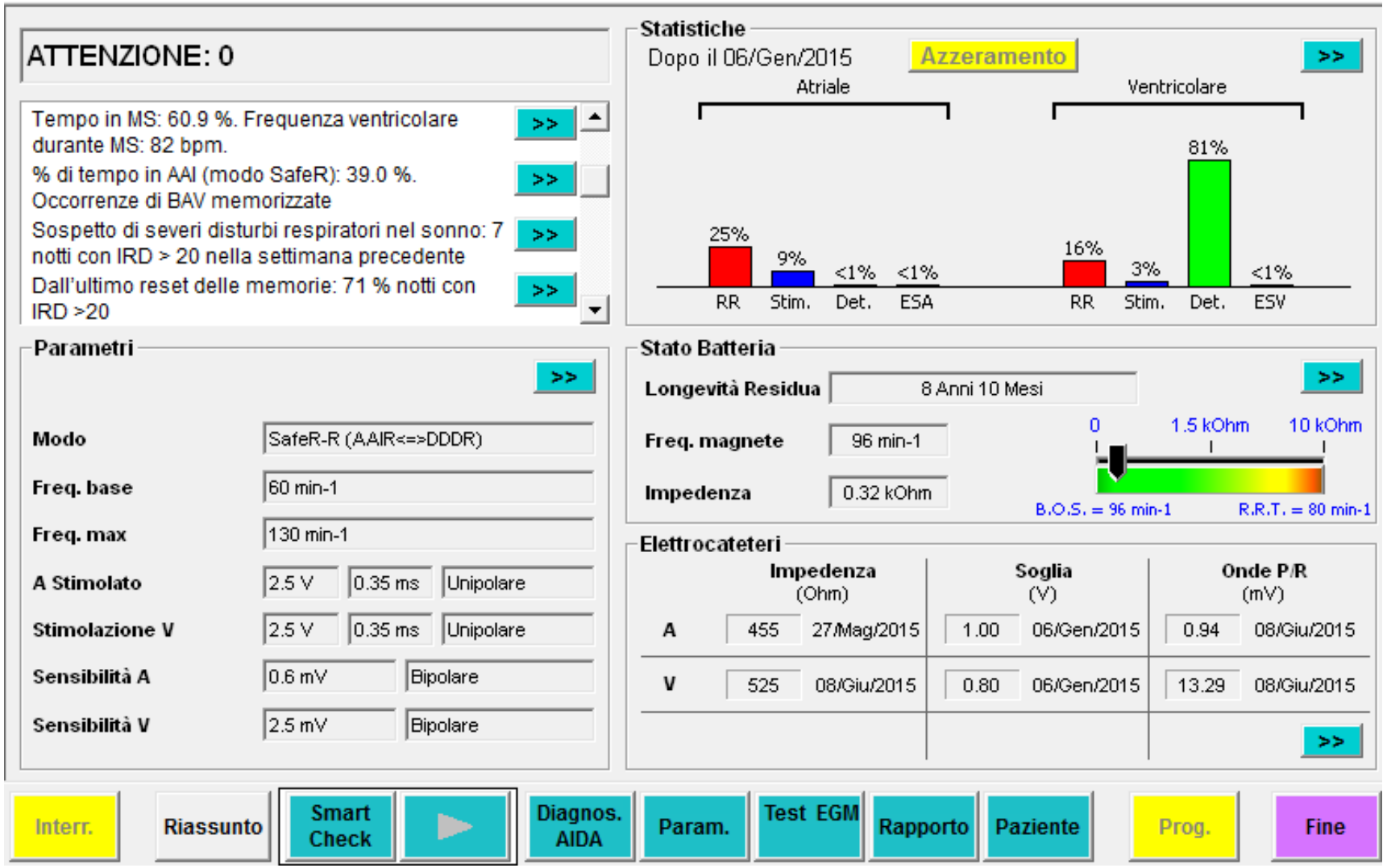
- Donna di 66 anni
- BMI=29.30 kg/m^2 (sovrappeso)
- Ipertesa, dislipidemica
- OSAS severa
  (AHI=45.1/h alla polisonnografia)



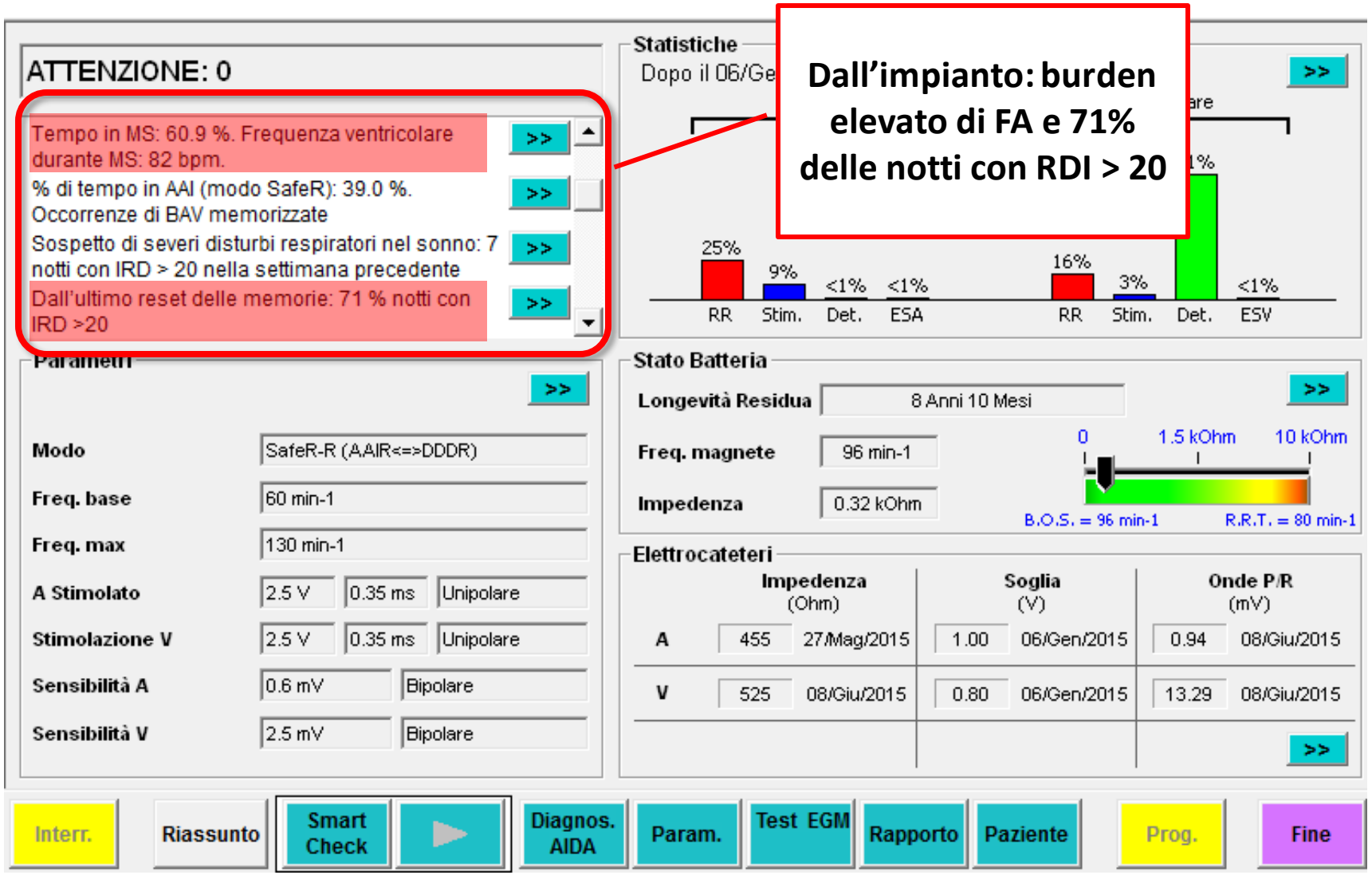
- Paziente in CPAP

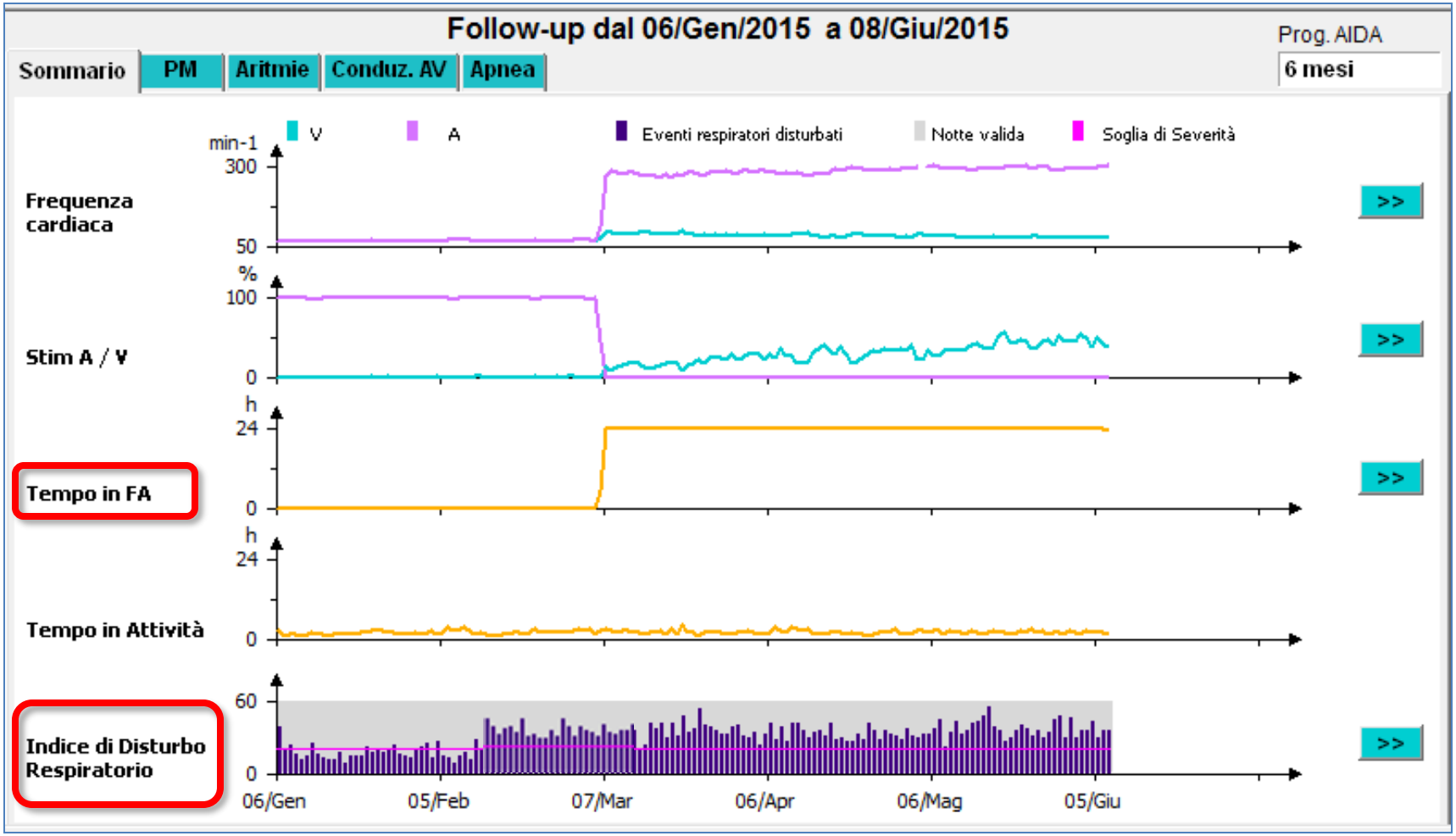
- PMK impiantato a Ottobre 2014 per Sindrome bradi-tachi

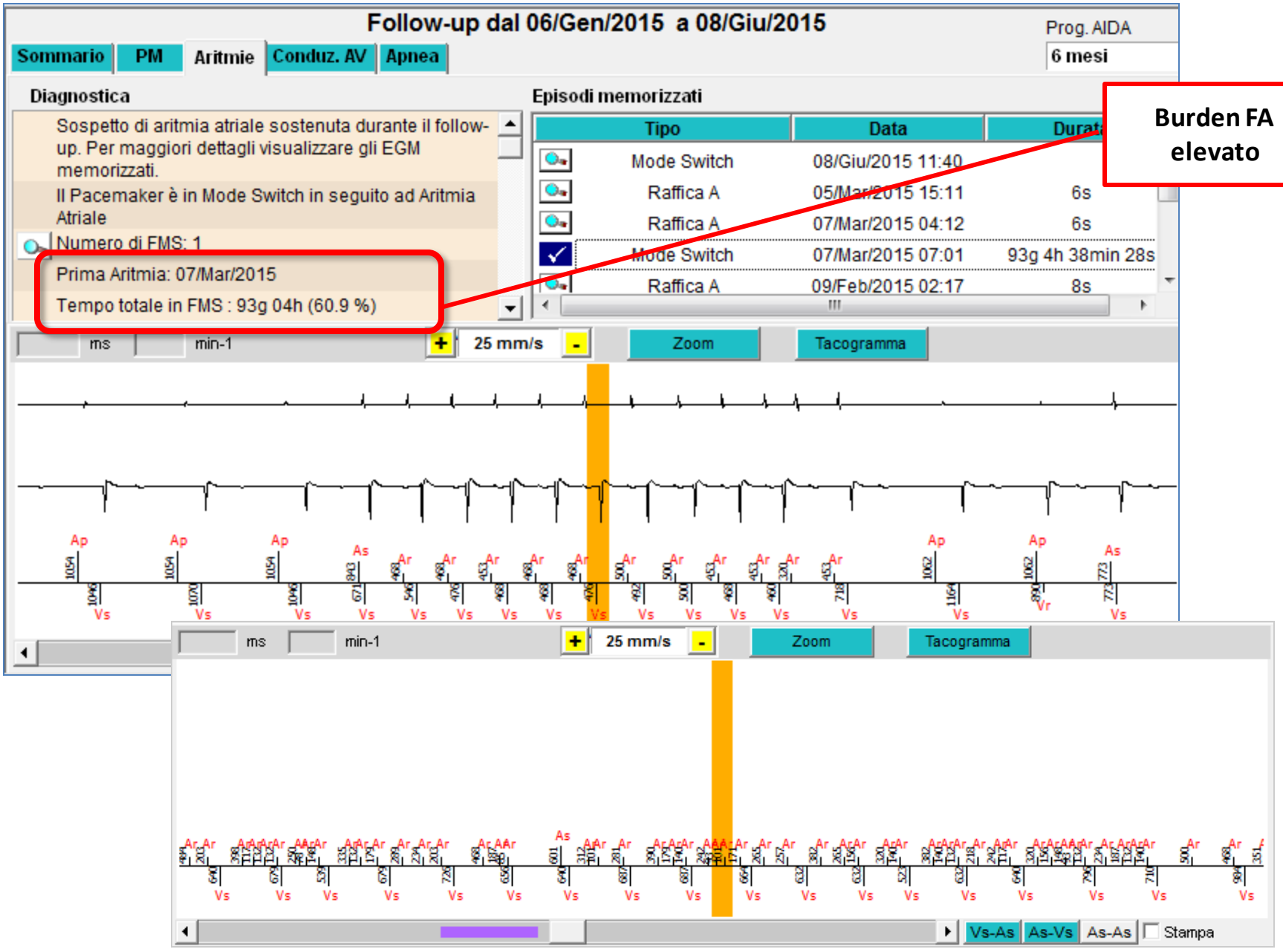
Follow-up 8 mesi dall'impianto, giugno 2015

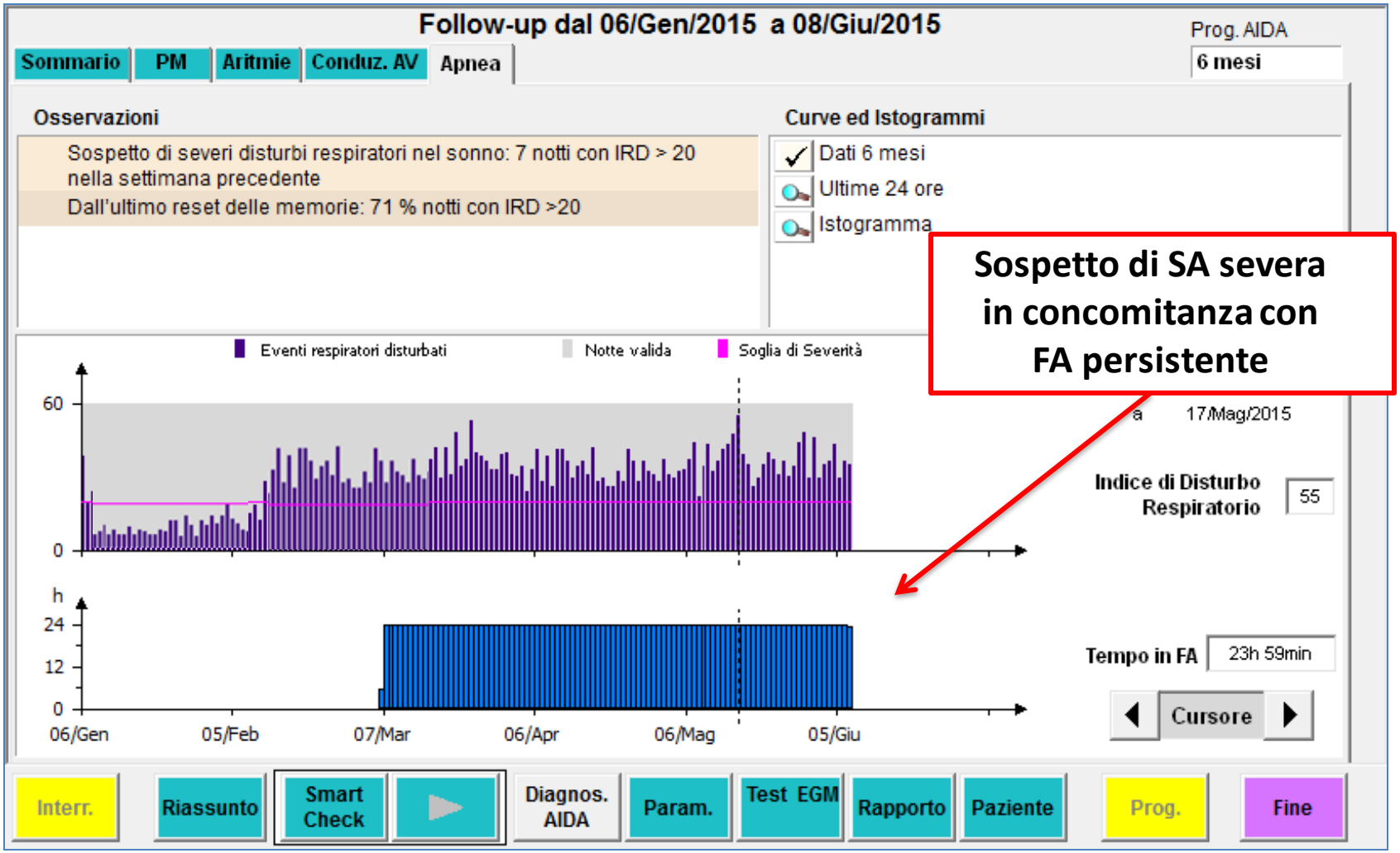


Follow-up 8 months later: June 8th, 2015

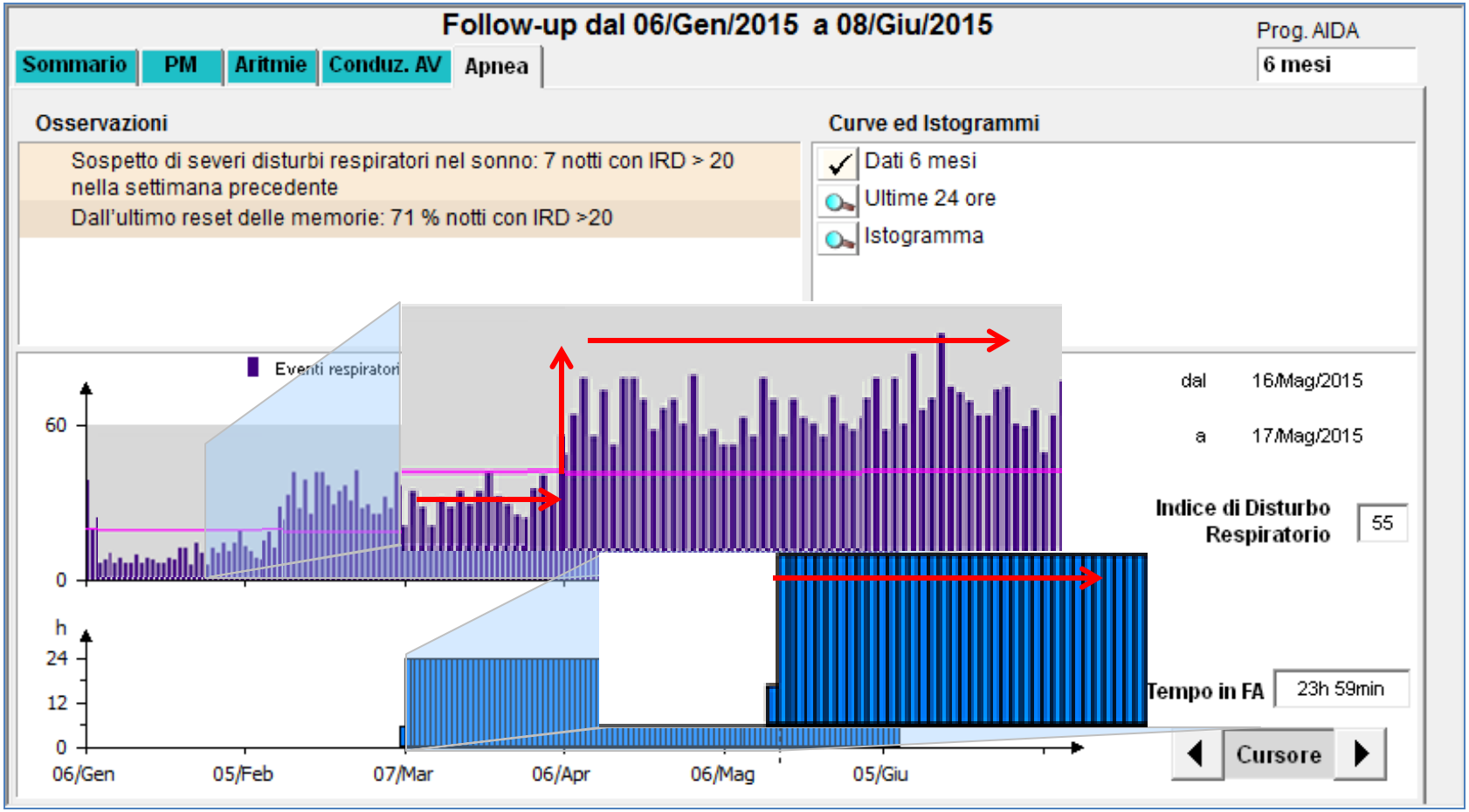



Follow-up dal 06/Gen/2015 a 08/Giu/2015
Prog. AIDA
6 mesi
Sommario
PM
Aritmie
Conduz. AV
Apnea
V
A
Eventi respiratori disturbati
Notte valida
Soglia di Severità
Frequenza cardiaca
min-1
300
50
Stim A / V
%
100
0
Tempo in FA
h
24
0
Tempo in Attività
h
24
0
Indice di Disturbo Respiratorio
60
0
>>
06/Gen
05/Feb
07/Mar
06/Apr
06/Mag
05/Giu


Follow-up dal 06/Gen/2015 a 08/Giu/2015
Prog. AIDA
6 mesi
Sommario
PM
Aritmie
Conduz. AV
Apnea
Diagnostica
Sospetto di aritmia atriale sostenuta durante il follow-up. Per maggiori dettagli visualizzare gli EGM memorizzati.
Il Pacemaker è in Mode Switch in seguito ad Aritmia Atriale
Numero di FMS: 1
Prima Aritmia: 07/Mar/2015
Tempo totale in FMS : 93g 04h (60.9 %)
Episodi memorizzati
Tipo
Data
Mode Switch 08/Giu/2015 11:40
Raffica A 05/Mar/2015 15:11 6s
Raffica A 07/Mar/2015 04:12 6s
Mode Switch 07/Mar/2015 07:01 93g 4h 38min 28s
Raffica A 09/Feb/2015 02:17 8s
Burden FA elevato
ms
min-1
25 mm/s
Zoom
Tacogramma
Vs-As
As-Vs
As-As
Stampa

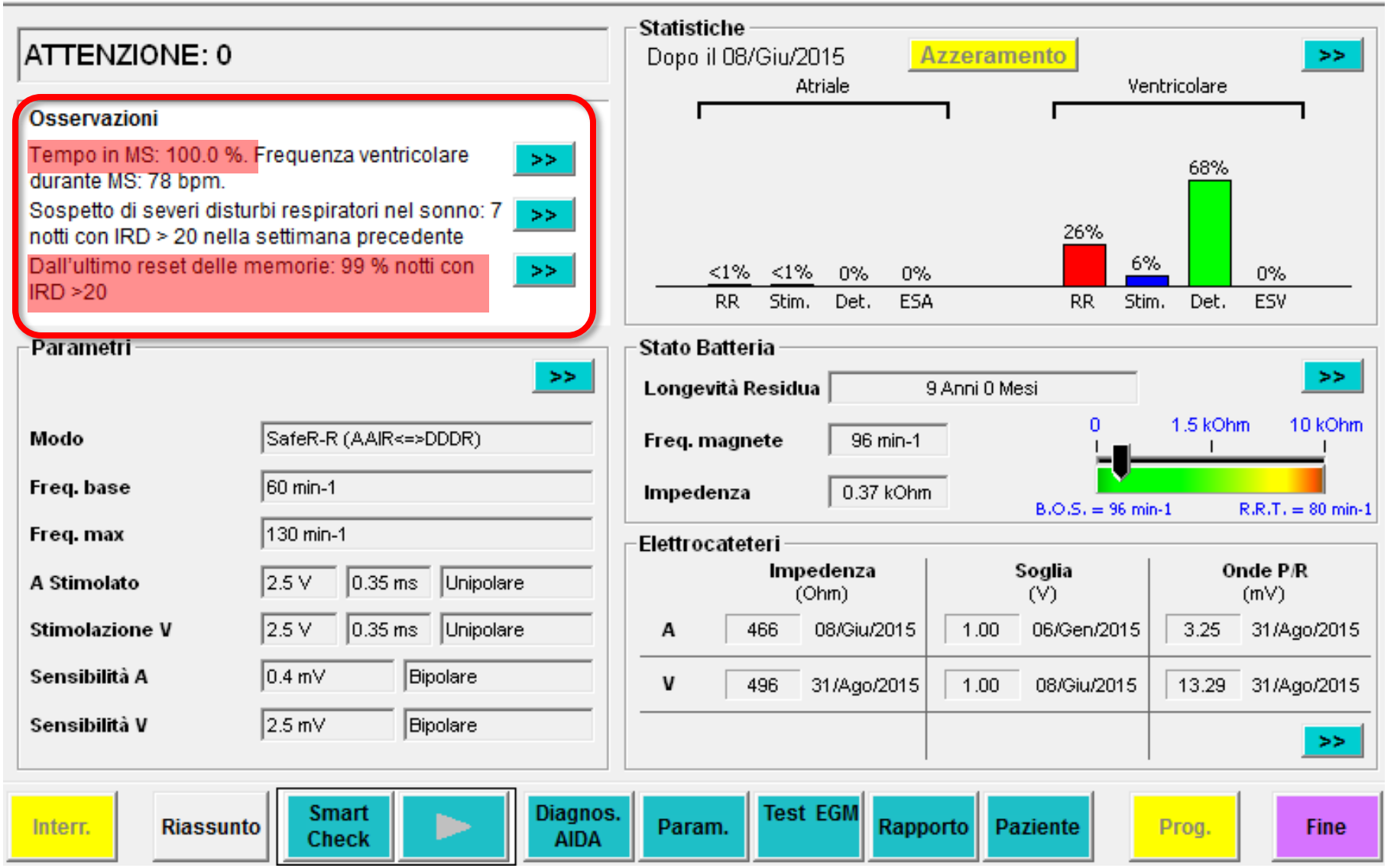

Follow-up dal 06/Gen/2015 a 08/Giu/2015
Prog. AIDA
6 mesi
Sommario
PM
Aritmie
Conduz. AV
Apnea
Osservazioni
Sospetto di severi disturbi respiratori nel sonno: 7 notti con IRD > 20 nella settimana precedente
Dall'ultimo reset delle memorie: 71 % notti con IRD >20
Curve ed Istogrammi
Dati 6 mesi
Ultime 24 ore
Istogramma
Sospetto di SA severa in concomitanza con FA persistente
Eventi respiratori disturbati
Notte valida
Soglia di Severità
60
0
a 17/Mag/2015
Indice di Disturbo Respiratorio
55
h
24
12
0
Tempo in FA
23h 59min
Cursore
06/Gen
05/Feb
07/Mar
06/Apr
06/Mag
05/Giu
Interr.
Riassunto
Smart Check
Diagnos. AIDA
Param.
Test EGM
Rapporto
Paziente
Prog.
Fine

Associazione FA e Sleep Apnea

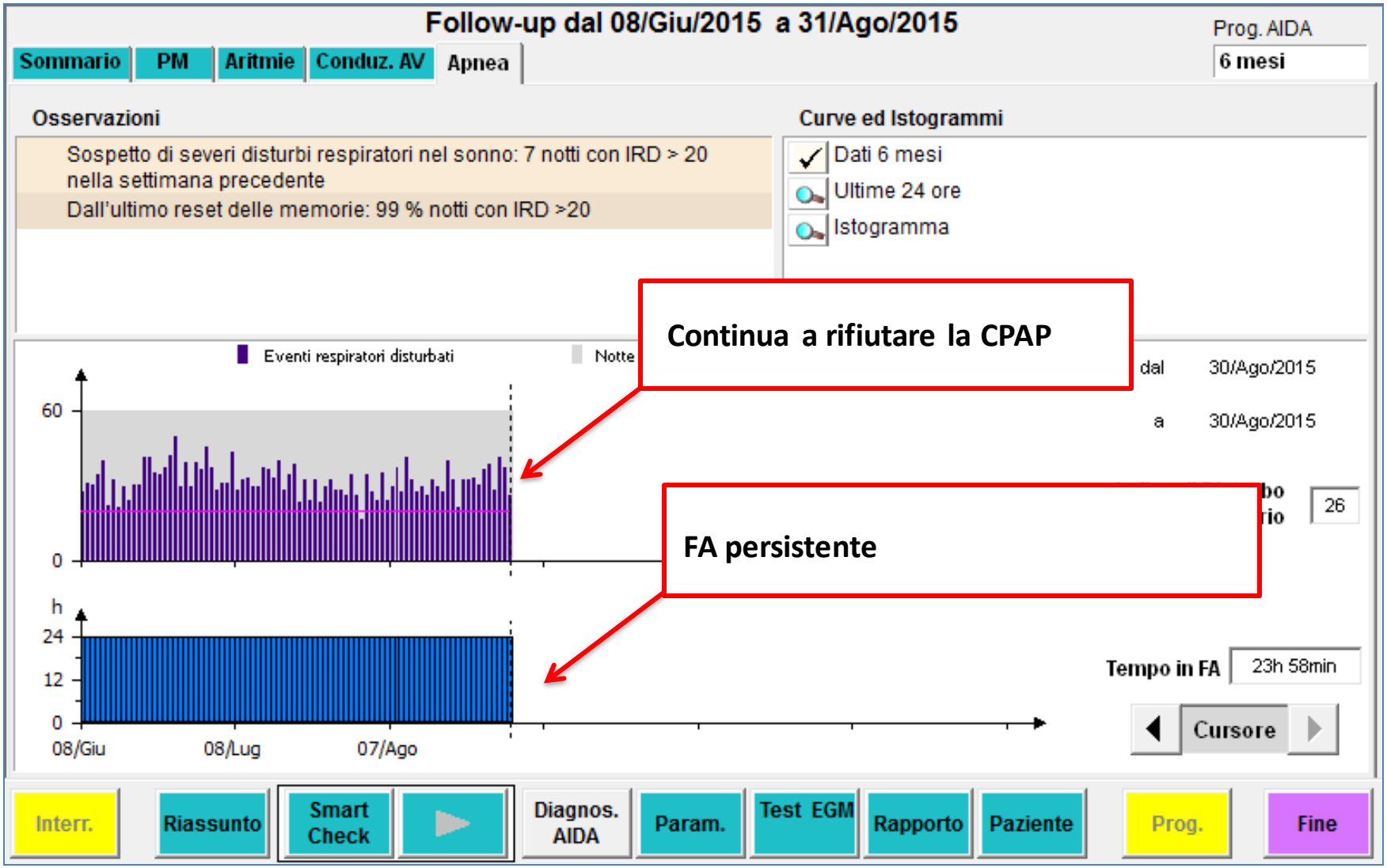


- buona compliance alla CPAP: RDI<20
- Ridotta compliance: aumento drammatico dell'RDI
- SA severa: trigger FA

Follow-up 31/08/2015

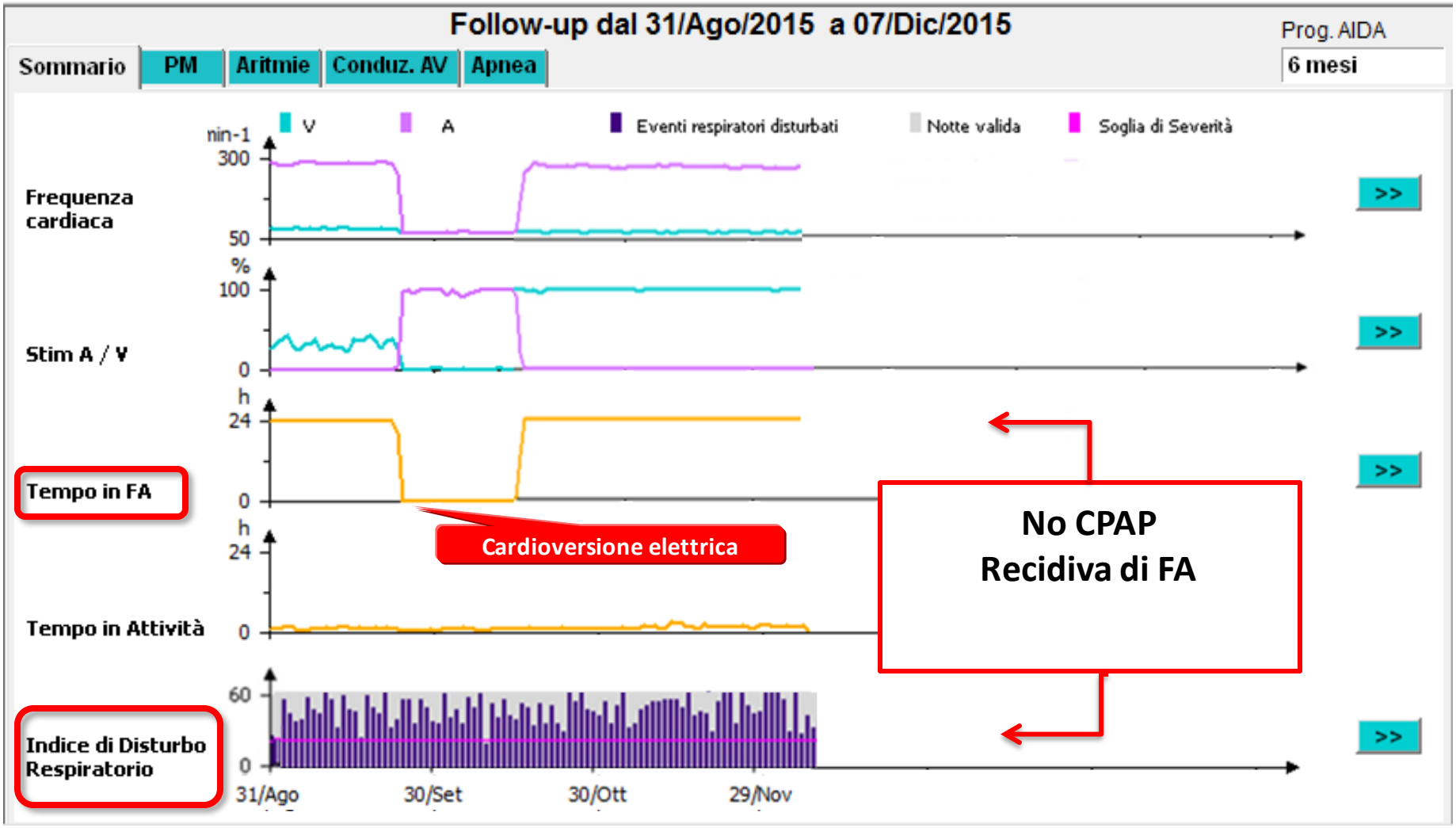


Correlation betweeen PM-RDI and AF



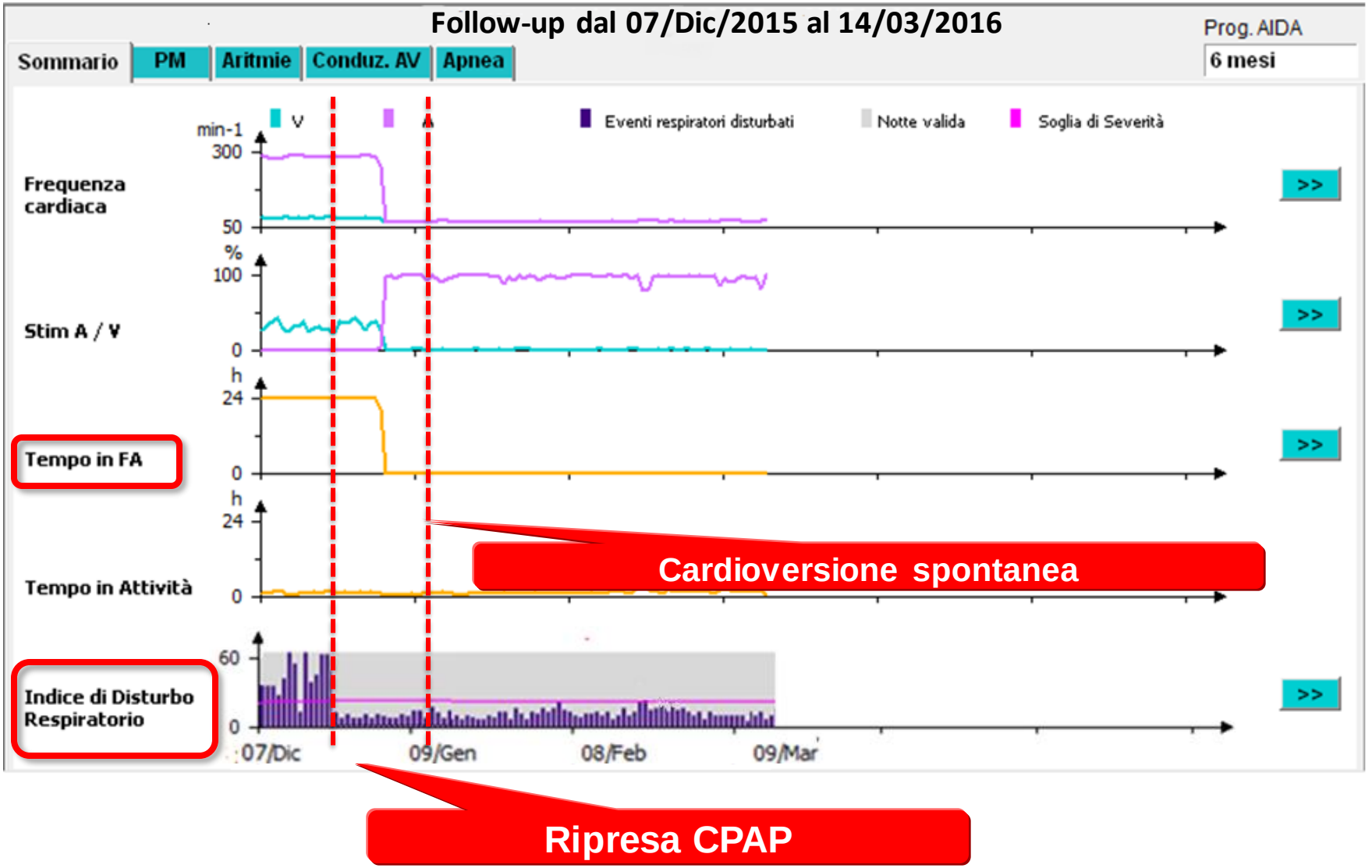
>> Performed an electrical cardioversion (Sept 2015)

Follow-up 07/12/2015 after cardioversion (Sep 2015)



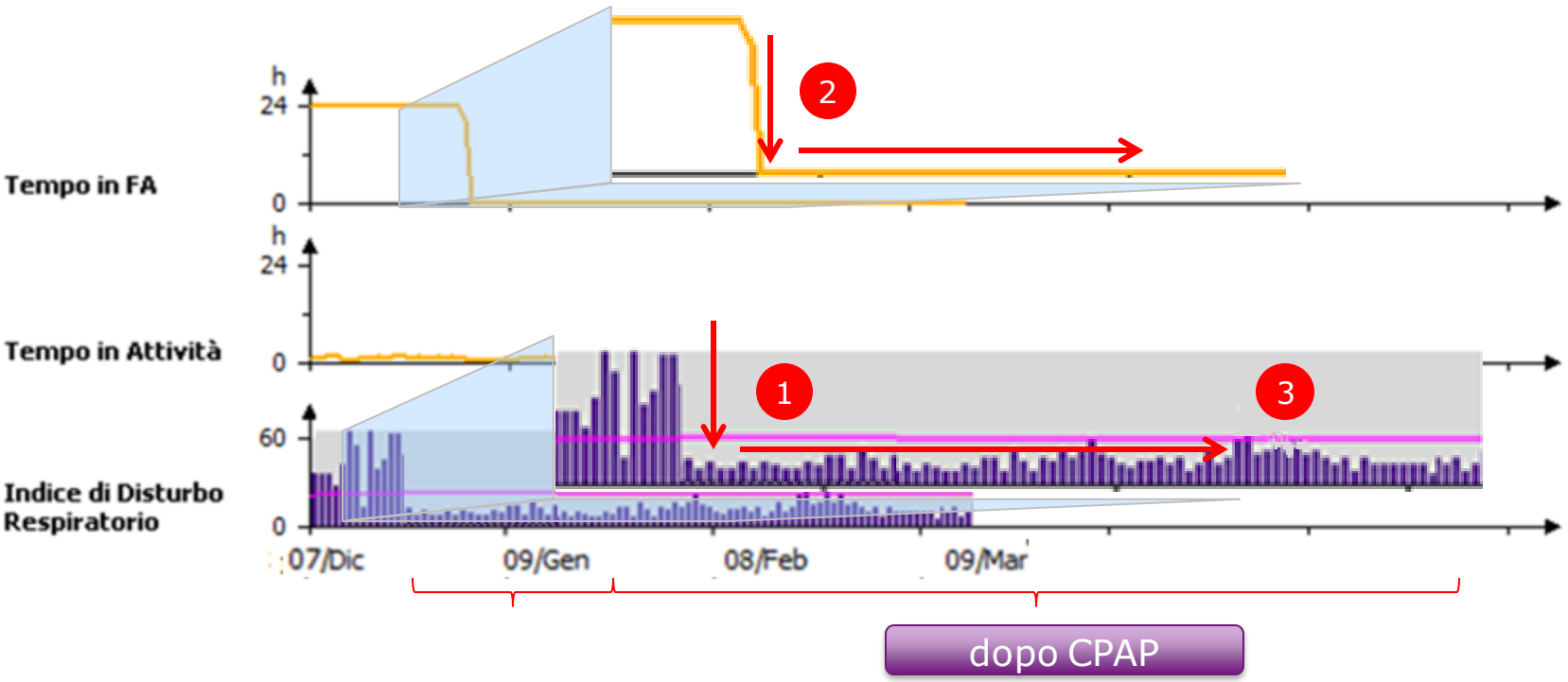
- Pz riferisce palpitazioni, sonnolenza durante il giorno, irritabilita', astenia
- Viene inviata allo pneumologo per trovare una soluzione e riprendere la CPAP

Follow-up 14/03/2016



PM-RDI e FA dopo la ripresa della CPAP

1. RDI si riduce drasticamente
2. Effetto positive sul ritmo (cardioversione spontanea)
3. Miglioramento della sintomatologia



Sleep Breathing Disorders

Effects of physiological cardiac pacing on sleep-disordered breathing in patients with chronic bradydysrhythmias

6 pts

Sex/age (years)	BMI (kg/m^2)	Heart disease	NYHA class	EF (%)	Mean HR (b.p.m.) Control	Mean HR (b.p.m.) PMI	AHI (/h) Control	AHI (/h) PMI	Lowest SaO_2 (%) Control	Lowest SaO_2 (%) PMI	CSB Control	CSB PMI
F/53	24.9	AVB	I	73	43	63	1.5	7.4	91	83	–	–
F/82	20.6	AVB	II	–	35	64	2.6	0.4	92	93	–	–
M/83	21.6	AVB	II	75	35	60	54.7	30.7	84	88	+	±
F/75	27.3	SND	II	70	40	60	20.6	11.4	65	67	+	–
F/67	26.1	SND	II	71	45	60	13.1	5.4	87	90	–	–
F/81	26.0	SND	II	75	40	60	0.8	3.6	90	89	–	–
74±12	24.4±2.7			73±2	40±4	61*±2	15.5±20.7	9.8±10.9	85±10	85±9	Mean±SD	

It seems that the increase in cardiac output after the pacemaker therapy resulted in a reduction of AHI and/or an improvement of Cheyne-Stokes breathing.

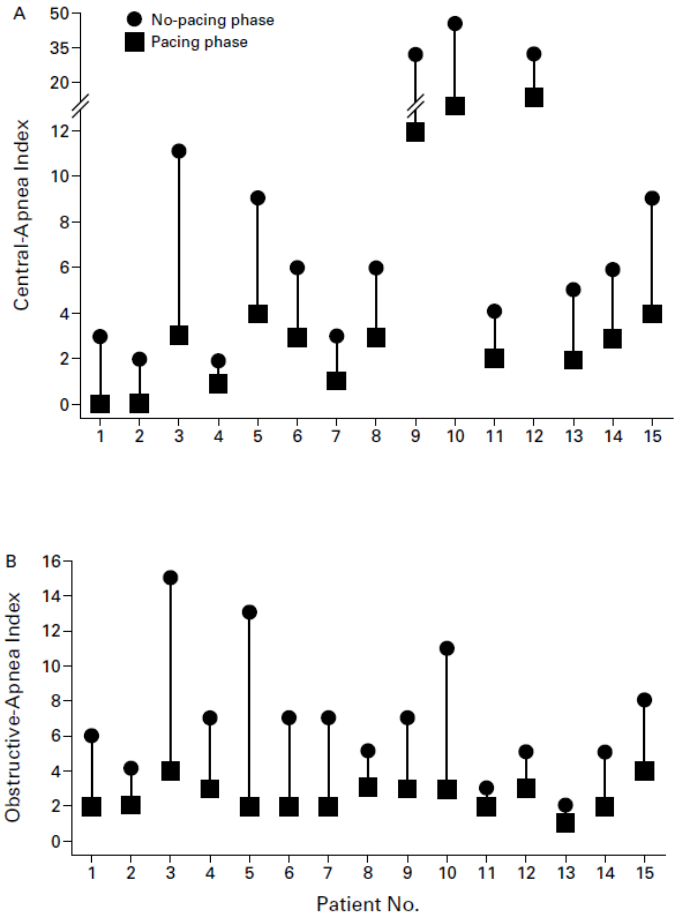
Kato et al. Psychiatry and Clinical Neurosciences 2001;55:257–258

The New England Journal of Medicine

BENEFIT OF ATRIAL PACING IN SLEEP APNEA SYNDROME

STEPHANE GARRIGUE, M.D., PHILIPPE BORDIER, M.D., PIERRE JAÏS, M.D., DIPEN C. SHAH, M.D., MELEZE HOCINI, M.D., CHANTAL RAHERISSON, M.D., MANUEL TUNON DE LARA, M.D., MICHEL HAÏSSAGUERRE, M.D., AND JACQUES CLEMENTY, M.D.

15 pts PM DDDR, tutti SAS+, cross-over randomizzato



Atrial overdrive pacing substantially reduced the number of episodes of central and obstructive apnea in patients with pacemakers that had previously been implanted for the treatment of the sick sinus syndrome or the bradycardia–tachycardia syndrome. Further studies are needed to elucidate the mechanisms involved in achieving these reductions and to assess the precise role of cardiac pacing in preventing symptoms, disability, and death in the general population of patients with sleep apnea syndrome.

In patients implanted with dual-chamber pacemakers for bradyarrhythmias, there is 60% reduction in both central and obstructive sleep apnoea severity by overdriving atrial pacing at 15 bpm faster than the mean baseline nocturnal cardiac frequency.

APNEA OSTRUTTIVA

Garrigue et al. NEJM 2002;346:404-412

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Atrial Overdrive Pacing for the Obstructive Sleep Apnea–Hypopnea Syndrome

16 pts

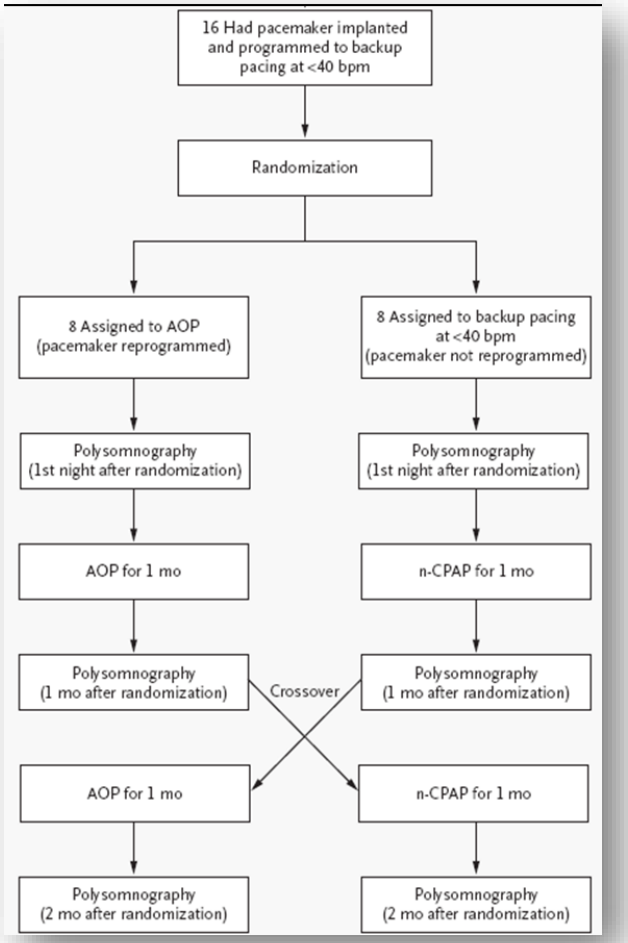


Table 3. Long-Term Effect of AOP and n-CPAP on Selected Variables.*

Intervention and Variable	Mean Baseline Value	Mean Value during Intervention	Difference between Baseline and Intervention Values	95% CI of Difference	P Value
AOP					
Apnea–hypopnea index†	49.0	49.2	+0.2	−2.7 to +2.3	0.87
Arousal index‡	40.75	41.3	+0.55	−2.0 to +3.1	0.65
Desaturation index§	56.1	56.4	+0.3	−2.6 to +3.2	0.82
Mean SaO_2 (%)	88.0	87.0	−1.0	−1.2 to +0.1	0.08
Lowest SaO_2 (%)	77.0	76.0	−1.0	−1.1 to +0.5	0.40
Total sleep time (min)	288.9	283.6	−5.3	−13.1 to +2.3	0.16
Duration of REM sleep (min)	10.1	8.2	−1.9	−3.9 to +0.2	0.07
PCO_2 (mm Hg)	39.3	39.4	+0.1	−0.4 to +0.5	0.75
Epworth Sleepiness Scale score¶	15.7	15.8	+0.1	−0.24 to +0.36	0.67
n-CPAP					
Apnea–hypopnea index†	49.0	2.7	−46.3	−56.2 to −36.5	<0.001
Arousal index‡	40.75	4.6	−36.15	−44.9 to −27.4	<0.001
Desaturation index§	56.1	3.5	−52.6	−64.8 to −40.4	<0.001
Mean SaO_2 (%)	88.0	96.0	+8.0	+5.7 to +9.3	<0.001
Lowest SaO_2 (%)	77.0	90.0	+13.0	+9.9 to +16.1	<0.001
Total sleep time (min)	288.9	288.3	−0.6	−11.5 to +10.1	0.89
Duration of REM sleep (min)	10.1	17.4	+7.3	+4.8 to +9.9	<0.001
PCO_2 (mm Hg)	39.3	38.1	−1.2	−1.6 to −0.8	<0.001
Epworth Sleepiness Scale score¶	15.7	5.5	−10.2	−11.7 to −8.8	<0.001

In conclusion, n-CPAP therapy was highly effective in the treatment of patients with obstructive sleep apnea, whereas AOP had no effect on a representative population of such pts.

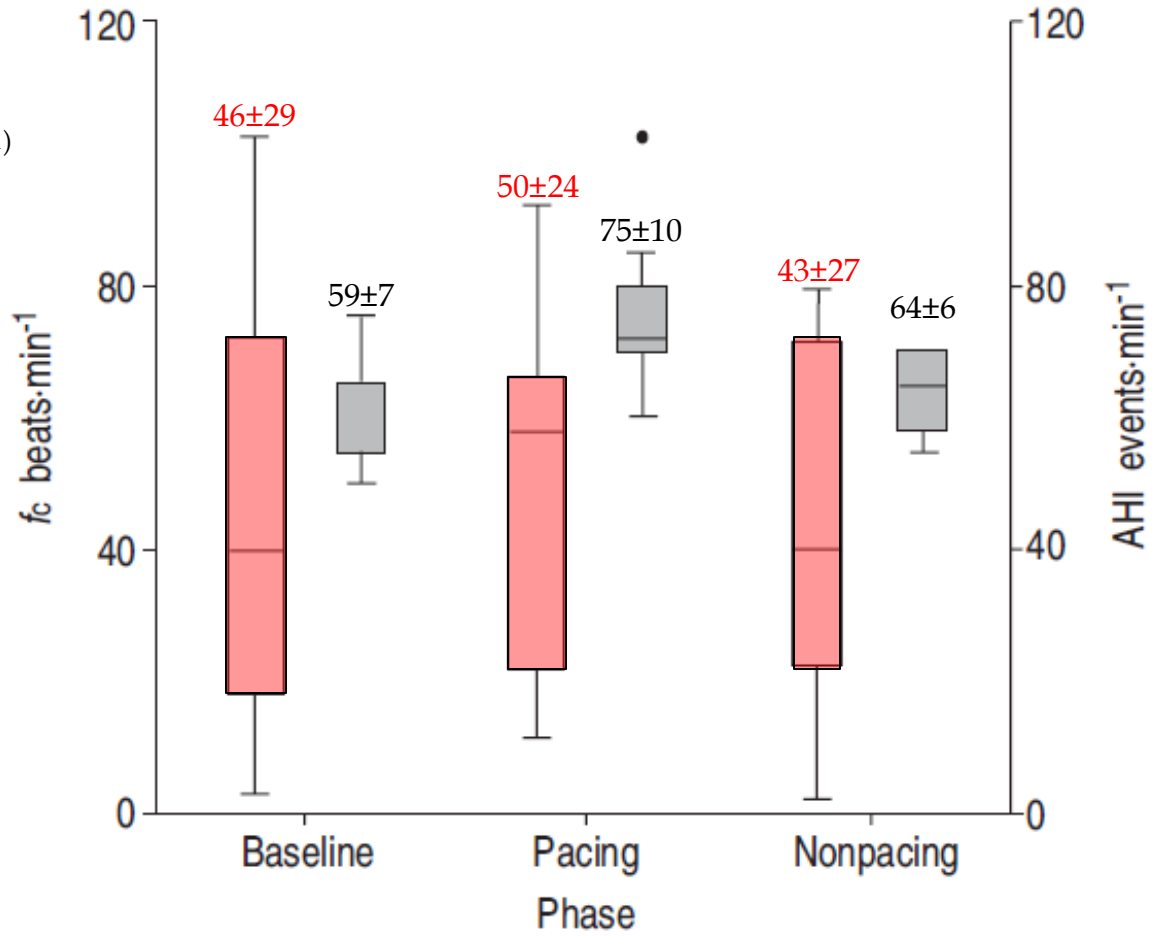
Simantirakis et al. NEJM 2005;353:2568-2577

Overdrive atrial pacing does not improve obstructive sleep apnoea syndrome

17 pts

fc (p<0.001)

AHI (p=NS)

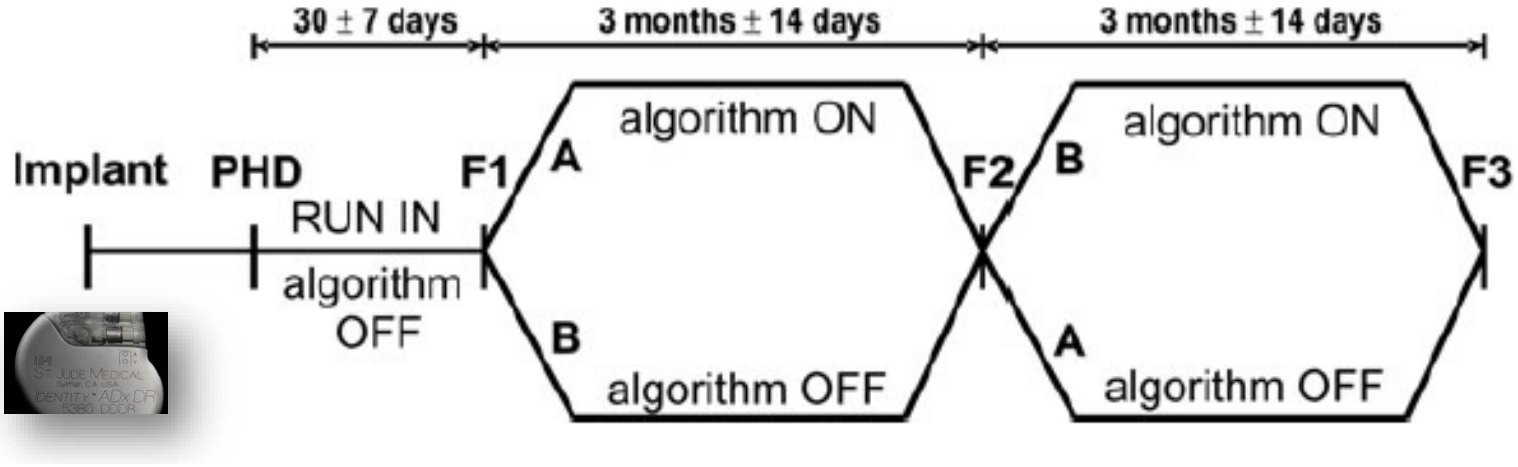


According to current knowledge, atrial overdrive pacing has no significant effect on obstructive sleep apnoea.

Pe´pin et al. Eur Respir J 2005; 25: 343–347

Long-Term Effects of Dynamic Atrial Overdrive Pacing on Sleep-Related Breathing Disorders in Pacemaker or Cardioverter Defibrillator Recipients

12 pts; **EF= 38.3 ± 13.6%;** obstructive or mixed SAS. All patients received Identity™ DR dual chamber pacemakers (St. Jude Medical) or Epic™+ DR or Atlas™+DR ICD (St. Jude Medical) with the AFx™ algorithm (AOP 5–10 bpm above the patient's intrinsic rhythm) and bipolar pacing leads compatible with these devices.



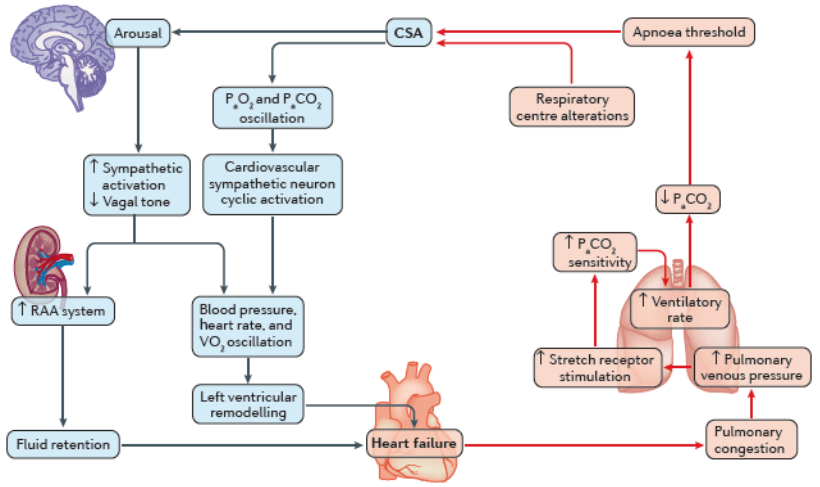
Measurement	Study Group	Baseline	4 months	7 months
Apnea-hypopnea index	All patients	38.0 ± 29.7	29.4 ± 17.9	36.2 ± 20.5
	Group A	26.5 ± 28.9	30.5 ± 23.8	21.5 ± 23.3
	Group B	43.8 ± 32.5	28.7 ± 15.4	43.5 ± 17.4***

In conclusion, the prevalence of obstructive or mixed SAS was high in patients with a depressed left ventricular ejection fraction and an indication for pacing or ICD therapy. In these patients, dynamic atrial overdrive pacing did not (1) alleviate SAS, (2) increase exercise capacity, or (3) decrease neuro-humoral activation, and, therefore, should not be offered as a therapeutic alternative to CPAP.

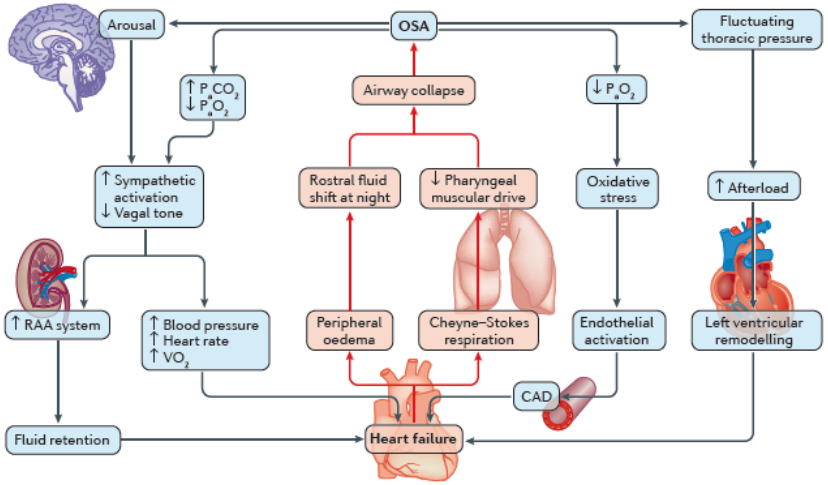
SINHA et al. PACE 2009; 32:S219–S222

Sleep-disordered breathing occurs in more than one-third of HF patients

- Mood disorders and psychological stress
- Medications: angiotensin-receptor blockers, loop diuretics, and β-blockers.
- Nocturia
- Both Central sleep apnoea, similar to Cheyne Stokes respiration, Obstructive sleep apnoea, and a mixed pattern of the two



CSA and OSA have been shown to be associated with a worse prognosis in HF.



Parati et al. NATURE REVIEWS | CARDIOLOGY 2016;13:389-403

REVIEW

Sleep apnoea in patients with heart failure: Part II: Therapy

Therapies with indirect effects on abnormal sleep-related respiratory events

When these therapies improve cardiac performance, they may alleviate sleep apnoea.

Medications for chronic congestive heart Failure

Nocturnal oxygen supplementation

Cardiac pacing

Philippe Bordier. Archives of Cardiovascular Disease 2009;102:711—720

Cardiac Resynchronization Therapy Improves Central Sleep Apnea and Cheyne-Stokes Respiration in Patients With Chronic Heart Failure

24 pts

		All Patients (n = 24)	CSA (n = 14)	No CSA (n = 10)	p Value (CSA vs. No CSA)
Cardiorespiratory polygraph					
Duration of recording (h)	Pre	8 ± 2	8 ± 2	8 ± 3	NS
	CRT	8 ± 3	8 ± 3	8 ± 2	NS
AHI (per h)	Pre	11.9 ± 11.7 (1–42)	19.2 ± 10.3 (9–42)	1.7 ± 0.7 (1–3)	0.00001
	CRT	3.3 ± 3.8* (0–12)	4.6 ± 4.4* (0–12)	1.5 ± 1.6 (0–4)	NS
SaO_2min (%)	Pre	88 ± 5 (70–92)	84 ± 5 (70–90)	90 ± 2 (88–92)	<0.05
	CRT	90 ± 2* (85–93)	89 ± 2* (85–90)	91 ± 1 (90–93)	NS
PSQI	Pre	7.5 ± 4.2 (2–14)	10.4 ± 1.6 (8–14)	2.4 ± 0.5 (2–4)	<0.05
	CRT	3.5 ± 2.2* (2–10)	3.9 ± 2.4* (2–10)	2.6 ± 0.9 (2–4)	NS

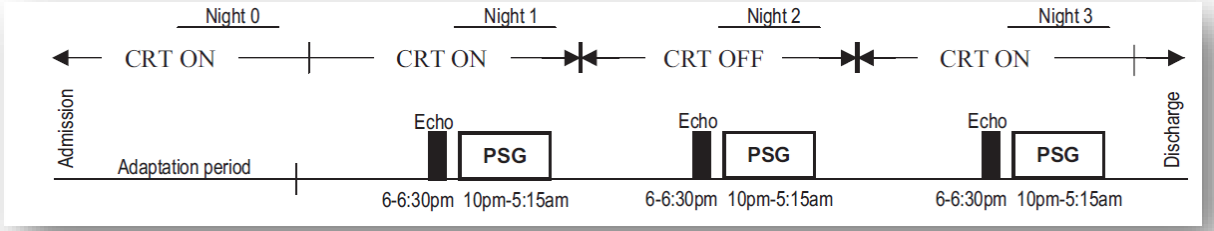
In patients receiving CRT, the improvement of cardiac function and exercise capacity is associated with a significant improvement of CSA with Cheyne-Stokes respiration and sleep quality.

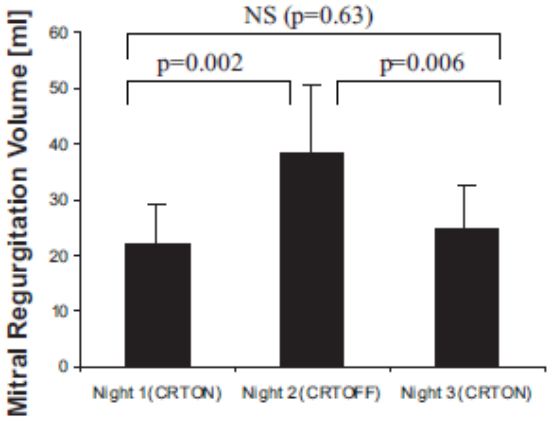
Sinha et al. JACC 2004;44:68-71

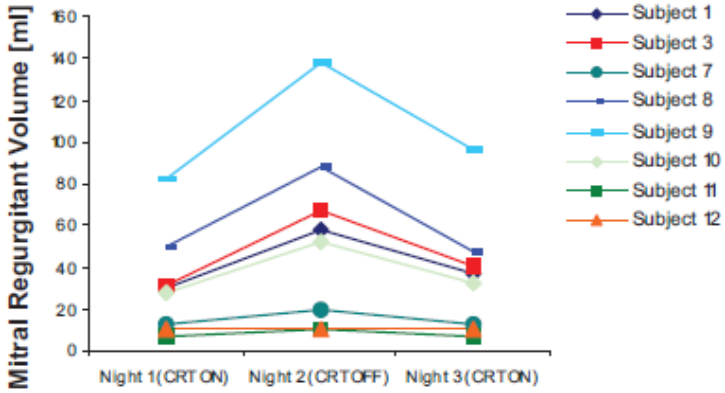
Short-term Effects of Cardiac Resynchronization Therapy on Sleep-Disordered Breathing in Patients With Systolic Heart Failure*

12 pts

Cardiomyopathy Type	QRS, ms	LVEF,† %
NDCM	160	15
NDCM	140	38
IDCM	160	28
NDCM	140	40
NDCM	160	19
NDCM	140	41
NDCM	160	25
IDCM	150	27
NDCM	160	13
NDCM	150	35
NDCM	180	34
IDCM	160	21
	150 ± 3.4	28 ± 2.8







CRT attenuates the severity of CSA in the short term in patients with advanced HF.

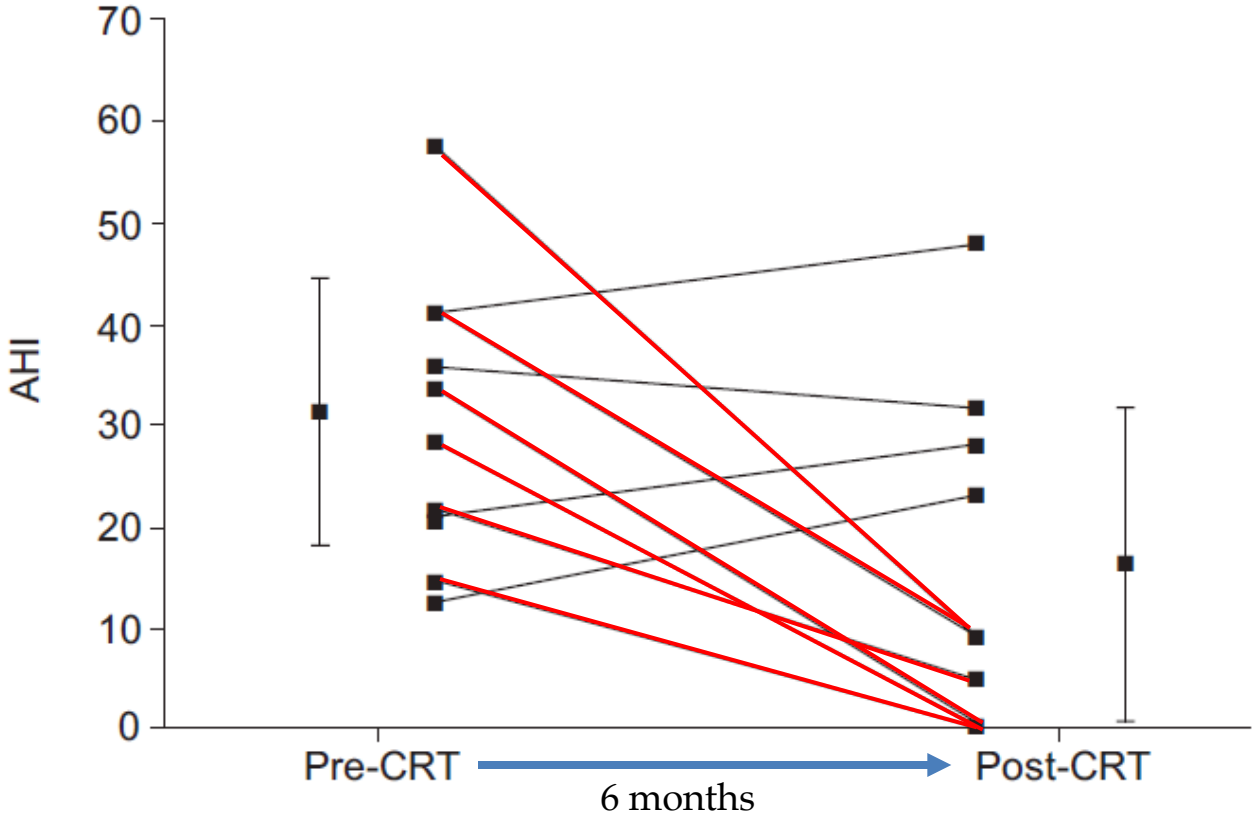
This attenuation may be linked to CRT-mediated reductions in mitral regurgitation severity.

CRT may be an important therapeutic option in patients with heart failure and CSA

Kara et al. CHEST 2008;134:87–93

Improvement in Cheyne-Stokes respiration following cardiac resynchronisation therapy

10 pts

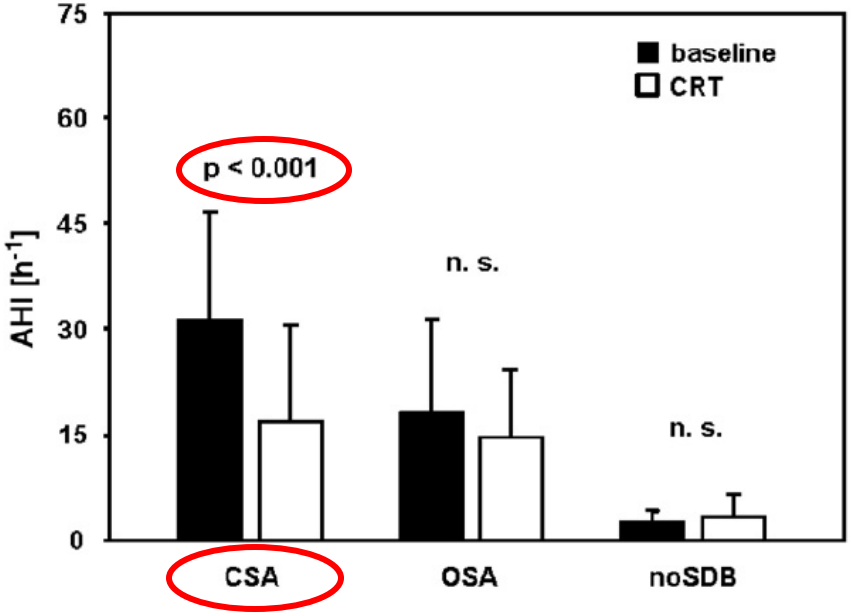


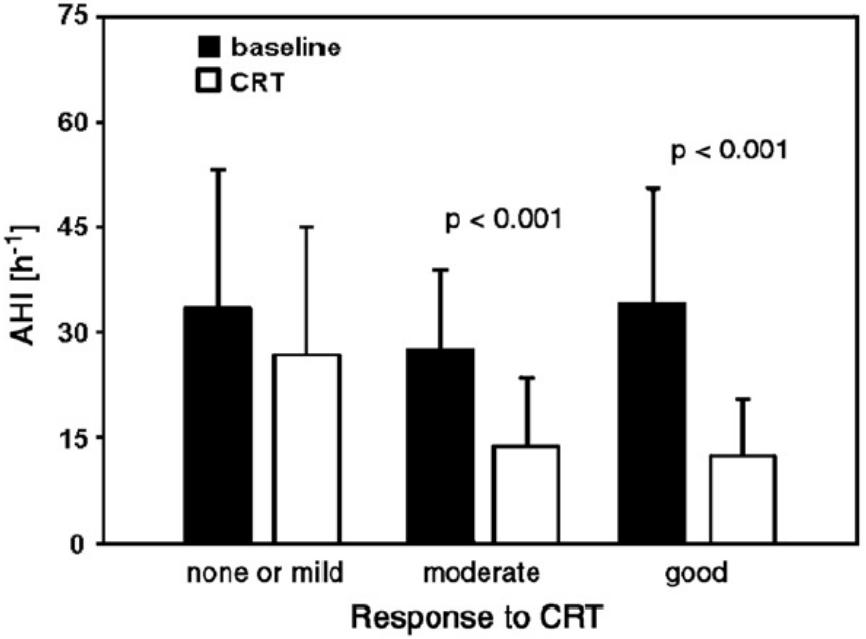
CRT is associated with a reduction in Cheyne-Stokes respiration, which may contribute to improved clinical outcome in patients treated with CRT

Gabor et al. Eur Respir J 2005; 26: 95–100

Influence of cardiac resynchronisation therapy on different types of sleep disordered breathing

77 pts: CSA 36; OSA 26; NoSDB

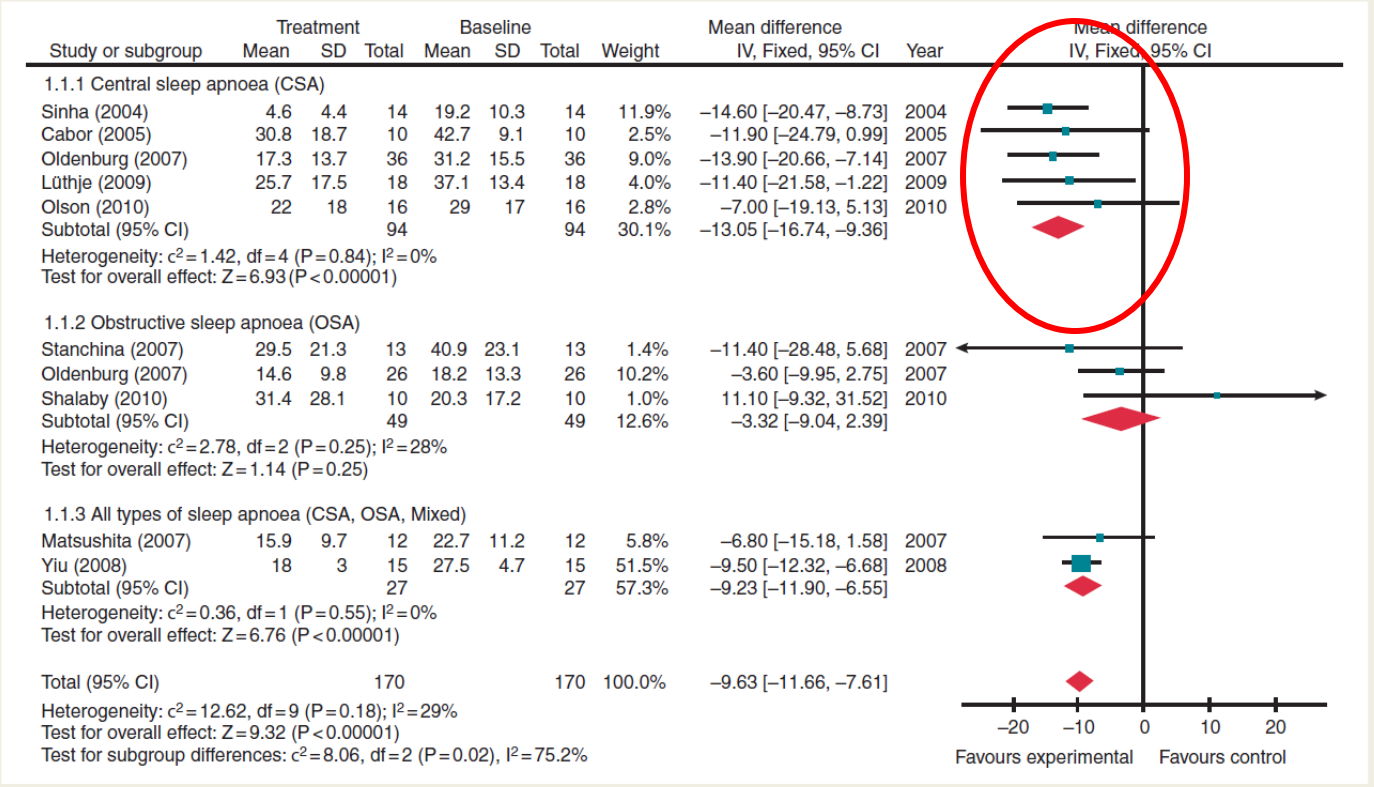




AHI improved only in patients with moderate or good response to CRT

Oldenburg et al. European Journal of Heart Failure 9 (2007) 820–826

Cardiac resynchronization therapy for the treatment of sleep apnoea: a meta-analysis



Study or subgroup	Treatment Mean	Treatment SD	Treatment Total	Baseline Mean	Baseline SD	Baseline Total	Weight	Mean difference IV, Fixed, 95% CI	Year
1.1.1 Central sleep apnoea (CSA)									
Sinha (2004)	4.6	4.4	14	19.2	10.3	14	11.9%	−14.60 [−20.47, −8.73]	2004
Cabor (2005)	30.8	18.7	10	42.7	9.1	10	2.5%	−11.90 [−24.79, 0.99]	2005
Oldenburg (2007)	17.3	13.7	36	31.2	15.5	36	9.0%	−13.90 [−20.66, −7.14]	2007
Lüthje (2009)	25.7	17.5	18	37.1	13.4	18	4.0%	−11.40 [−21.58, −1.22]	2009
Olson (2010)	22	18	16	29	17	16	2.8%	−7.00 [−19.13, 5.13]	2010
Subtotal (95% CI)			94			94	30.1%	−13.05 [−16.74, −9.36]	
Heterogeneity: $c^2 = 1.42$, df = 4 (P = 0.84); $I^2 = 0\%$									
Test for overall effect: Z = 6.93 (P < 0.00001)									
1.1.2 Obstructive sleep apnoea (OSA)									
Stanchina (2007)	29.5	21.3	13	40.9	23.1	13	1.4%	−11.40 [−28.48, 5.68]	2007
Oldenburg (2007)	14.6	9.8	26	18.2	13.3	26	10.2%	−3.60 [−9.95, 2.75]	2007
Shalaby (2010)	31.4	28.1	10	20.3	17.2	10	1.0%	11.10 [−9.32, 31.52]	2010
Subtotal (95% CI)			49			49	12.6%	−3.32 [−9.04, 2.39]	
Heterogeneity: $c^2 = 2.78$, df = 2 (P = 0.25); $I^2 = 28\%$									
Test for overall effect: Z = 1.14 (P = 0.25)									
1.1.3 All types of sleep apnoea (CSA, OSA, Mixed)									
Matsushita (2007)	15.9	9.7	12	22.7	11.2	12	5.8%	−6.80 [−15.18, 1.58]	2007
Yiu (2008)	18	3	15	27.5	4.7	15	51.5%	−9.50 [−12.32, −6.68]	2008
Subtotal (95% CI)			27			27	57.3%	−9.23 [−11.90, −6.55]	
Heterogeneity: $c^2 = 0.36$, df = 1 (P = 0.55); $I^2 = 0\%$									
Test for overall effect: Z = 6.76 (P < 0.00001)									
Total (95% CI)			170			170	100.0%	−9.63 [−11.66, −7.61]	
Heterogeneity: $c^2 = 12.62$, df = 9 (P = 0.18); $I^2 = 29\%$									
Test for overall effect: Z = 9.32 (P < 0.00001)									
Test for subgroup differences: $c^2 = 8.06$, df = 2 (P = 0.02), $I^2 = 75.2\%$									

Figure 2 Reduction of sleep apnoea parameters with the use of cardiac resynchronization therapy. CI, confidence intervals; IV, inverse variance; SD, standard deviation.

Lamba et al. Europace 2011;13,1174–1179

Conclusioni

- Elavata incidenza di SA nei portatori di device, associazione tra SA e FA: l'utilizzo degli algoritmi specifici descritti sembra un ottimo strumento di screening
- Il monitoraggio giornaliero e a lungo termine è utile per la gestione dell'SA e potrebbe impattare sulla prevenzione Cardiovascolare
- L'atrial overdrive pacing può ridurre l' AHI in una popolazione selezionata di pazienti
- Nei responders alla CRT si osserva una riduzione significativa dell'AHI specialmente nei pazienti con SA severa di tipo centrale