Ventriküler Erken Vurularda Olgu Bazlı Yaklaşım

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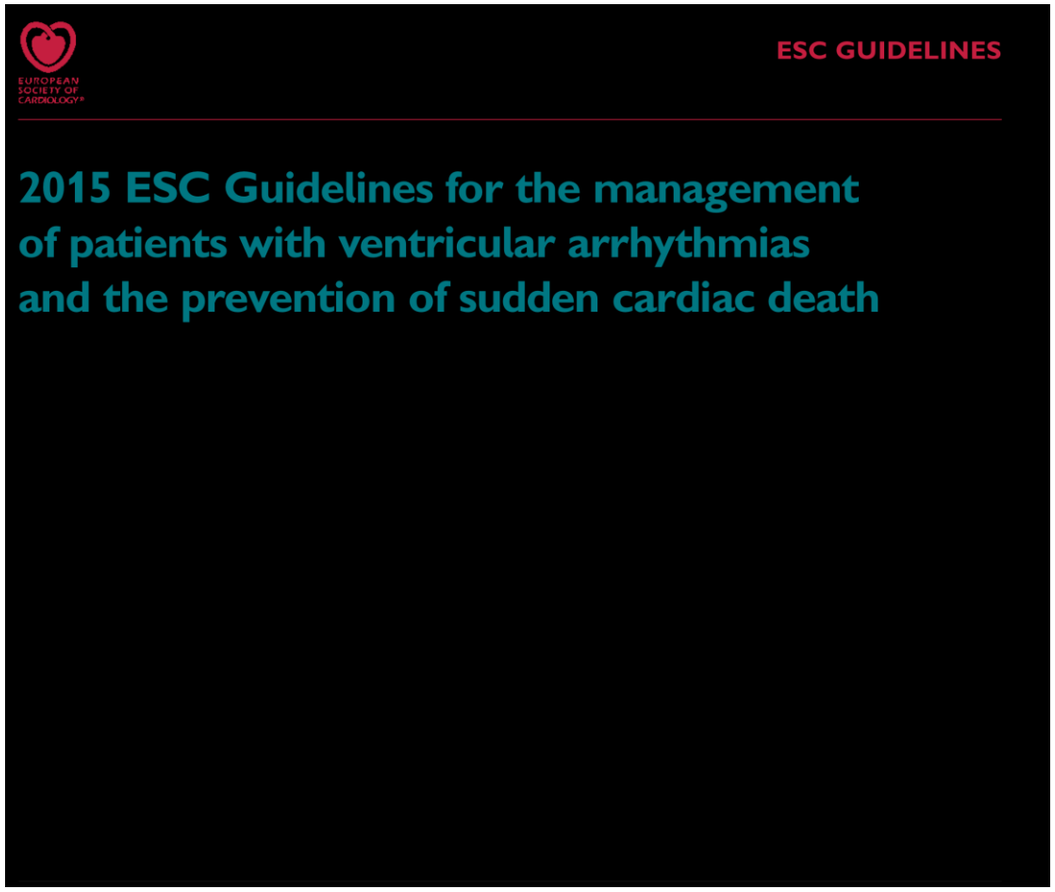
Acıbadem Maslak Hastanesi

Prematüre Ventriküler Vurularda Tedavi



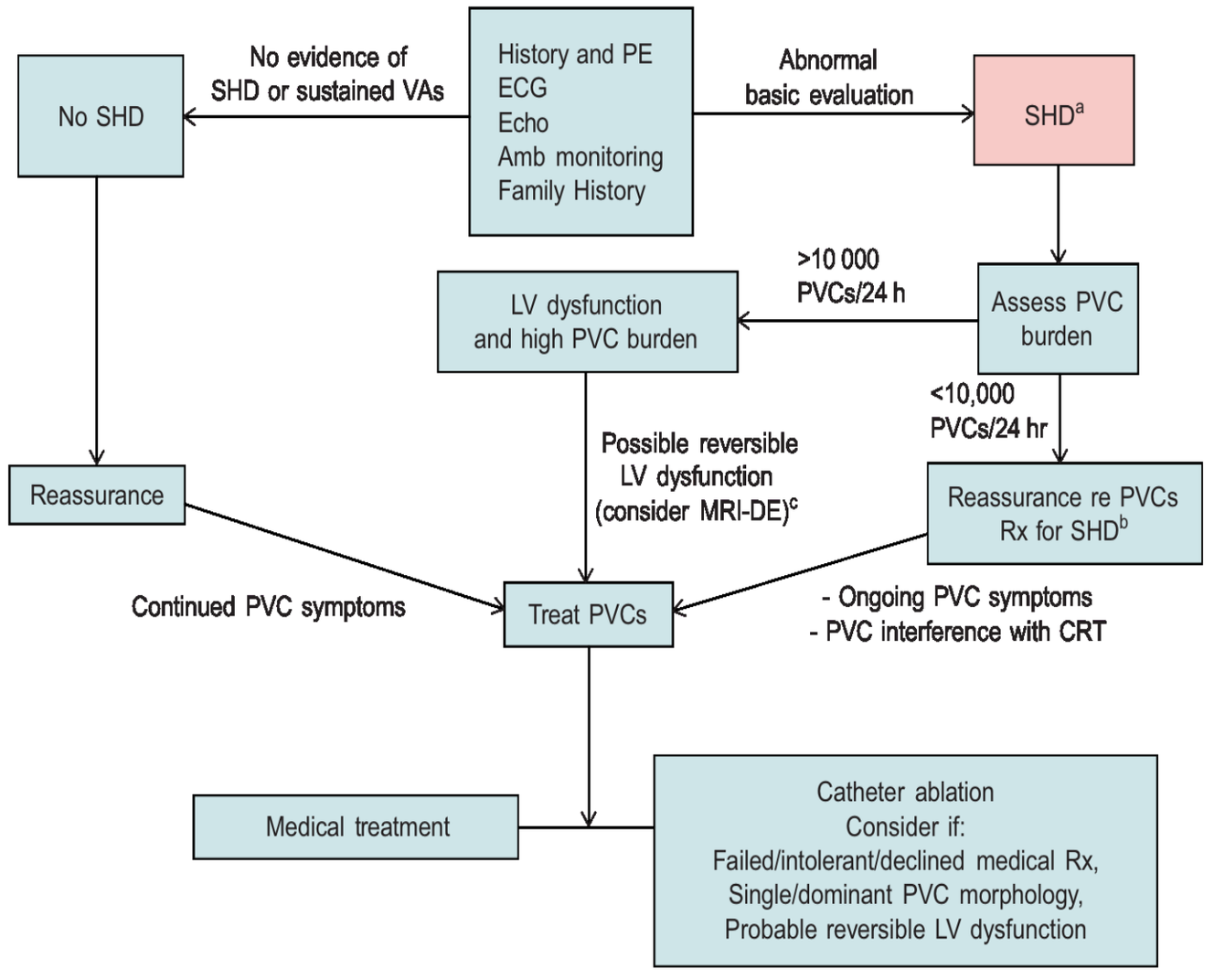


Figure 1 Management of PVCs. a. See table for definitions of structural heart disease; b. Medical therapy + ICD; c. Absence of high scar burden suggests reversibility. CRT = cardiac resynchronisation therapy; ICD = implantable cardioverter-defibrillator; LV = left ventricular; MRI-DE = magnetic resonance imaging with delayed enhancement; PE = physical examination; PVC = premature ventricular complexes; Rx = therapy; SHD = structural heart disease; VAs = ventricular arrhythmias.

Treatment of outflow tract ventricular tachycardia

Recommendations	Class[a]	Level[b]	Ref.[c]
Catheter ablation of RVOT VT/PVC is recommended in symptomatic patients and/or in patients with a failure of anti-arrhythmic drug therapy (e.g. beta-blocker) or in patients with a decline in LV function due to RVOT-PVC burden.	I	B	525–528
Treatment with sodium channel blockers (class IC agents) is recommended in LVOT/aortic cusp/epicardial VT/PVC symptomatic patients.	I	C	529–531
Catheter ablation of LVOT/aortic cusp/epicardial VT/PVC by experienced operators after failure of one or more sodium channel blockers (class IC agents) or in patients not wanting long-term anti-arrhythmic drug therapy should be considered in symptomatic patients.	IIa	B	195, 531–533

Olgu

- 16 y, Bayan
- 2 yıldır çarpıntı, göğüs ağrısı ve çarpıntı sırasında başdönmesi yakınması mevcut.
- EKG: NSR, VEA'lar (RBBB morfolojisi,inferior aks)
- Ekokardiyografi: Normal
- Kardiyak MR: Normal
- Egzersiz testi: Normal

ID:
Male

For N. KOHDEN FQW 110.2.140

Name:

Birth Date:

Years

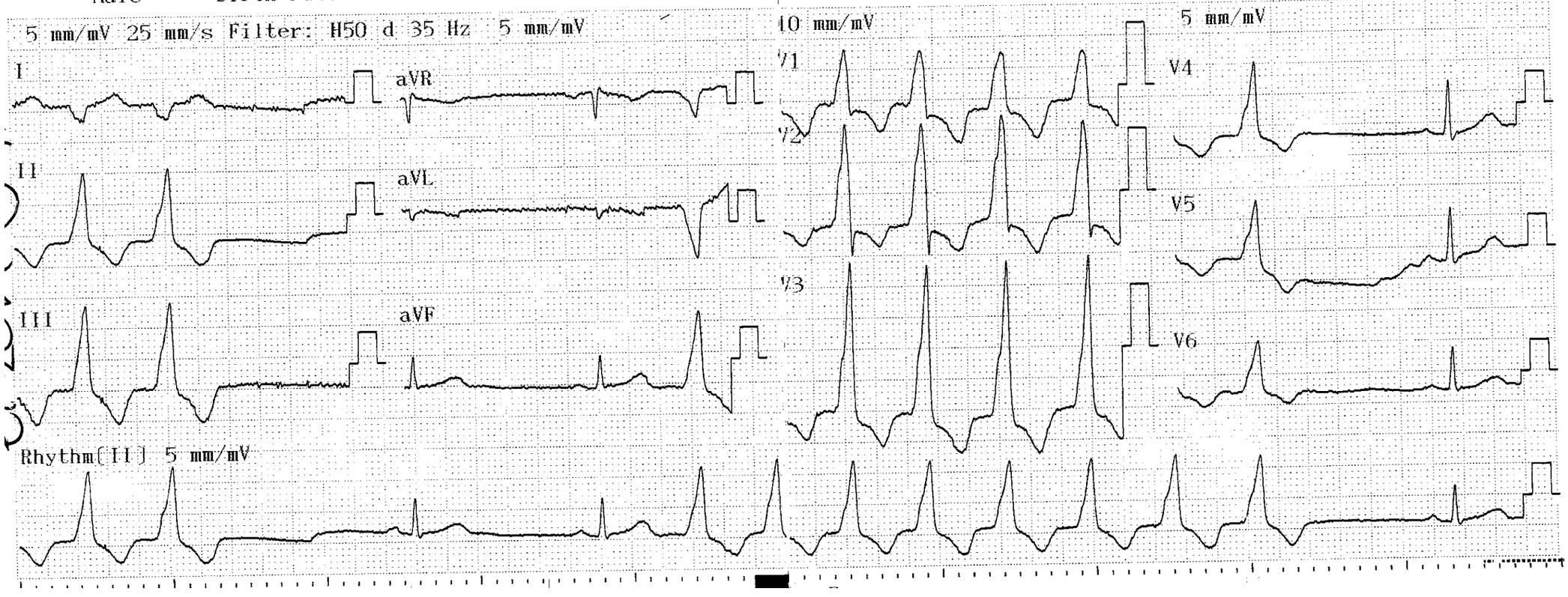
Medication:

cm kg mmHg

For N. KOHDEN FQW 110.2.140

Nov 30 2017 8:46 AM

114 bpm



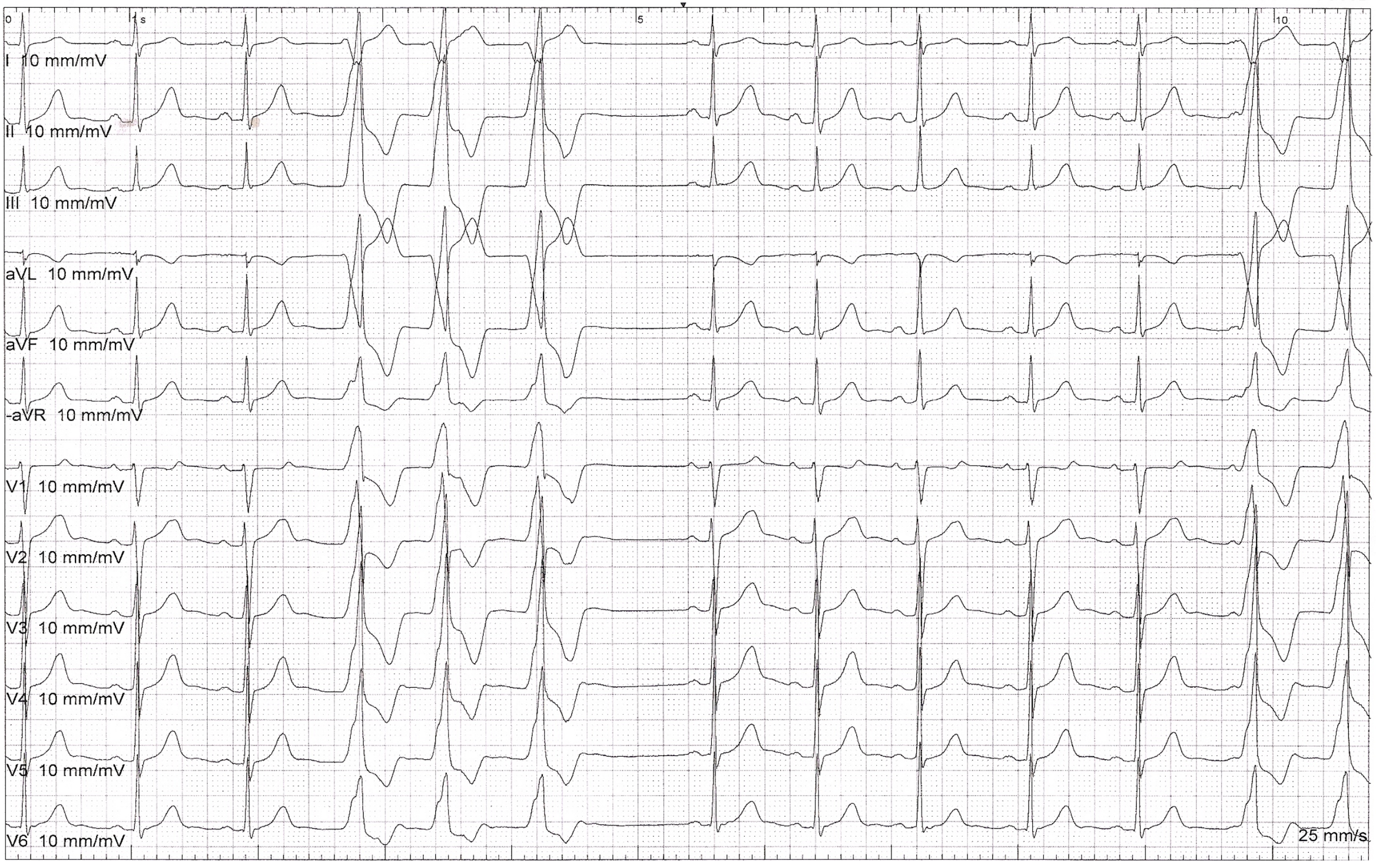
Olgu

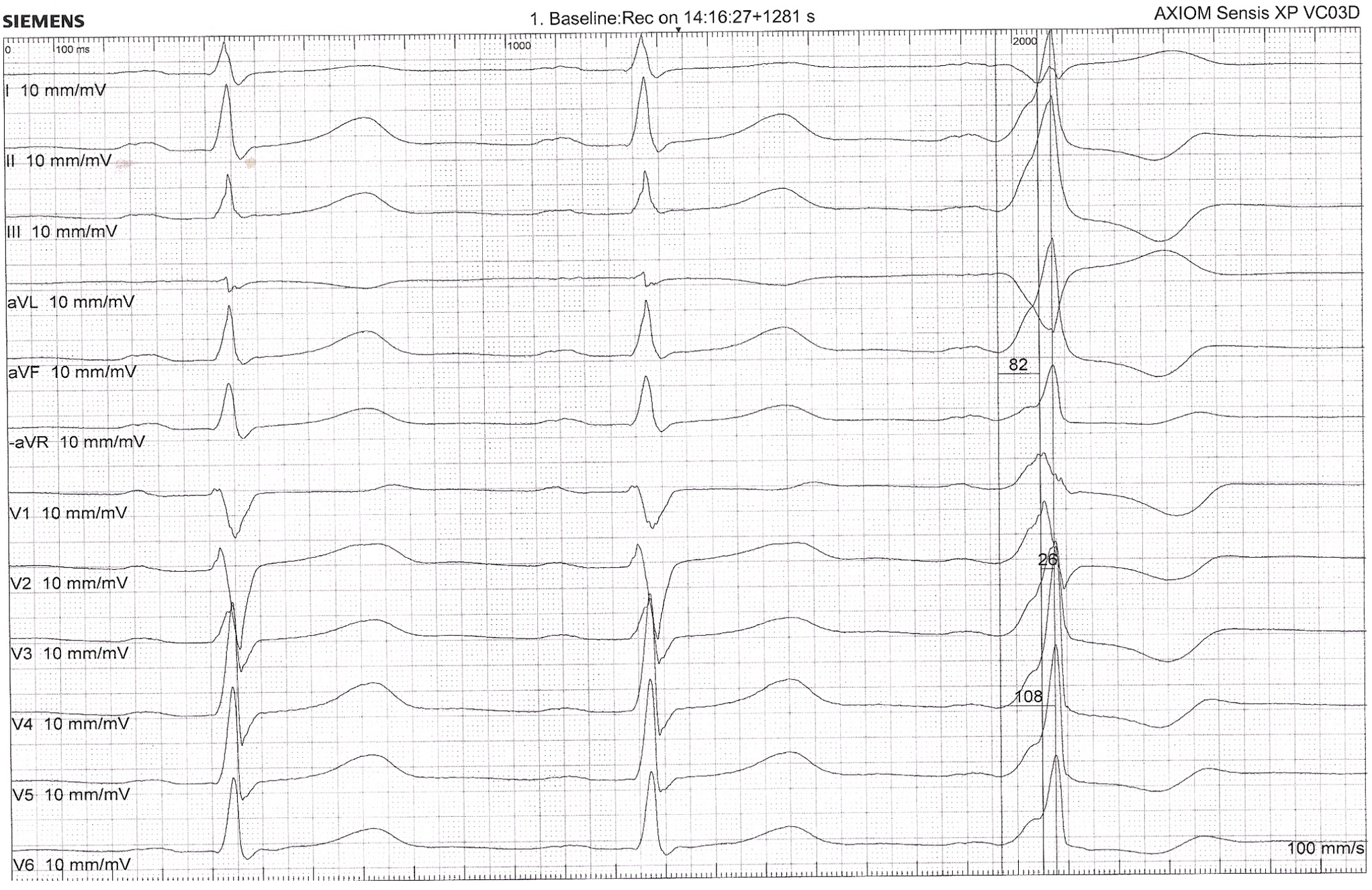
- Şikayetler ilaca dirençli;
  Bisoprolol
  Metoprolol
  Metoprolol+Sotalol
  Amiodarone
- 1.merkez:EFÇ yapılmış. VT uyarılamamış. Dual AV nodal fizyoloji saptanmış. Ablasyon yapılmamış.
- VT-Aortik kusp kökenli olduğu düşünülmüş
- 2.merkez: EFÇ, sol aortik kusp kökenli VT, kriyoablasyon uygulanmış.
- Nüks; bunun üzerine epikardiyal olarak aort kökünden ablasyon programlanmış. Ancak işlem sırasında komplikasyon?; sonrasında infeksiyon ve postperikardiyektomi sendromu gelişmiş.

SIEMENS

1. Baseline:Rec on 14:16:27+924 s

AXIOM Sensis XP VC03D




SIEMENS
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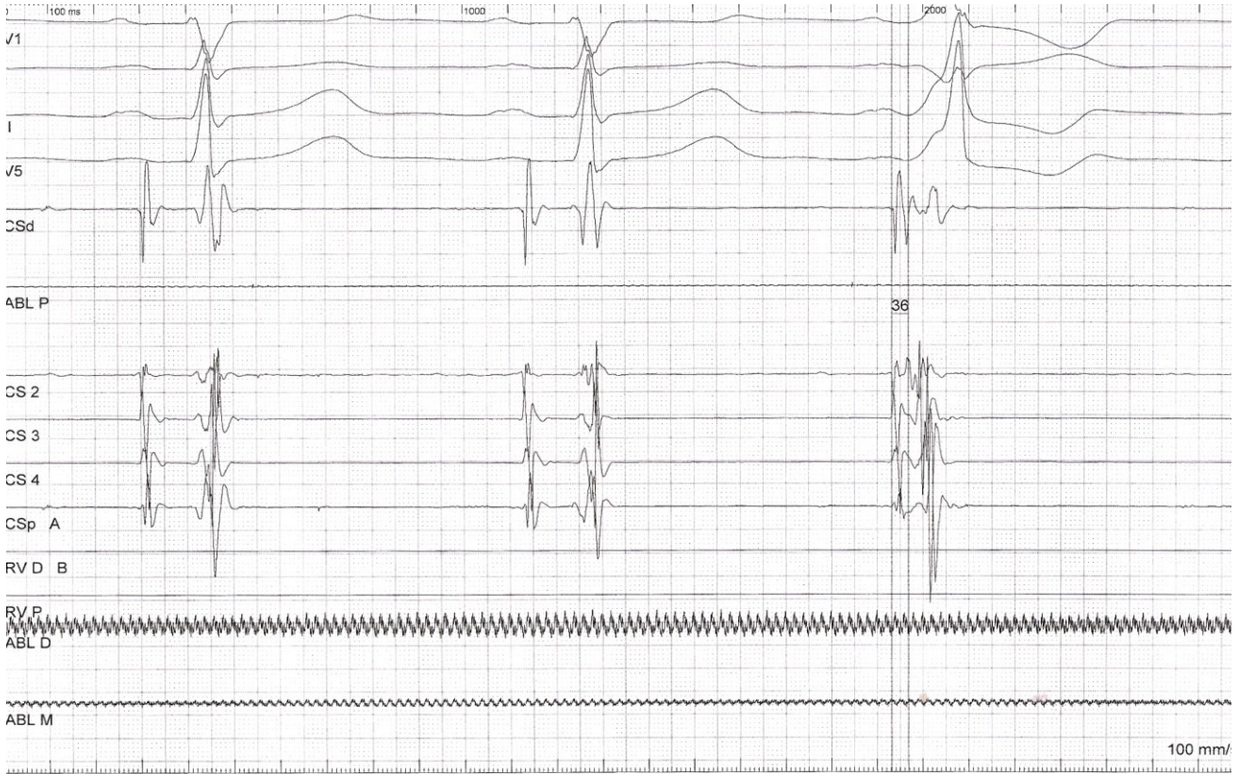
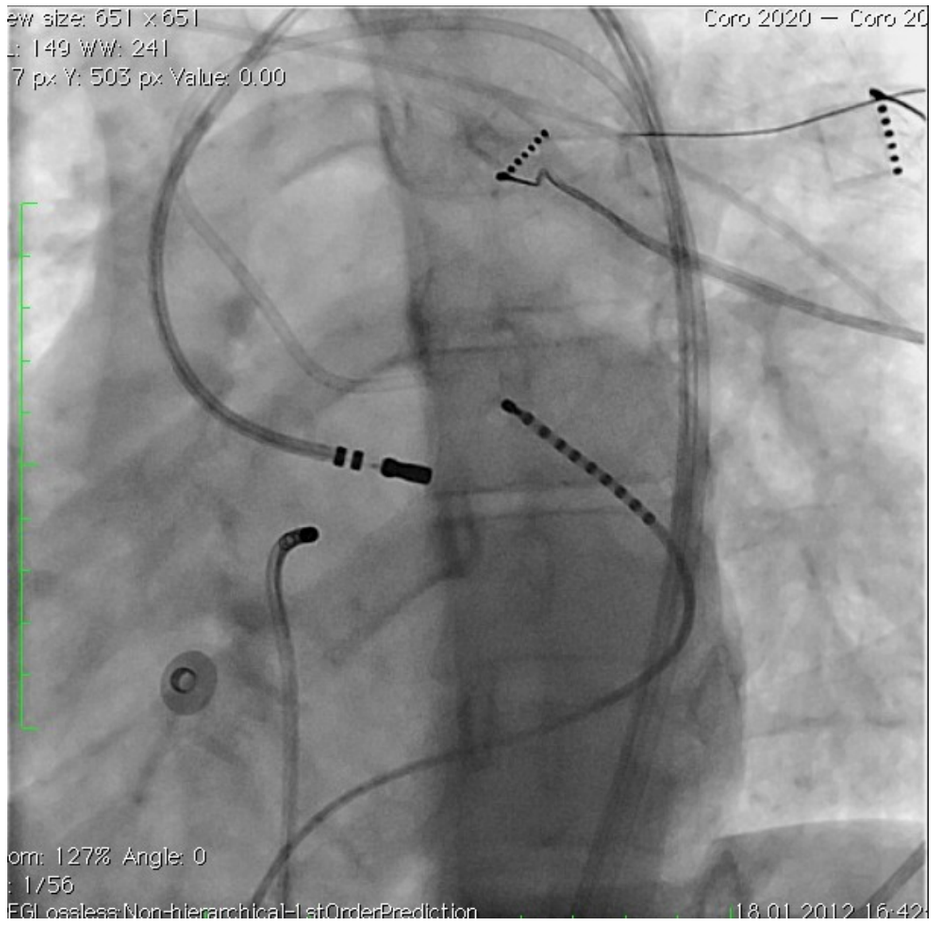
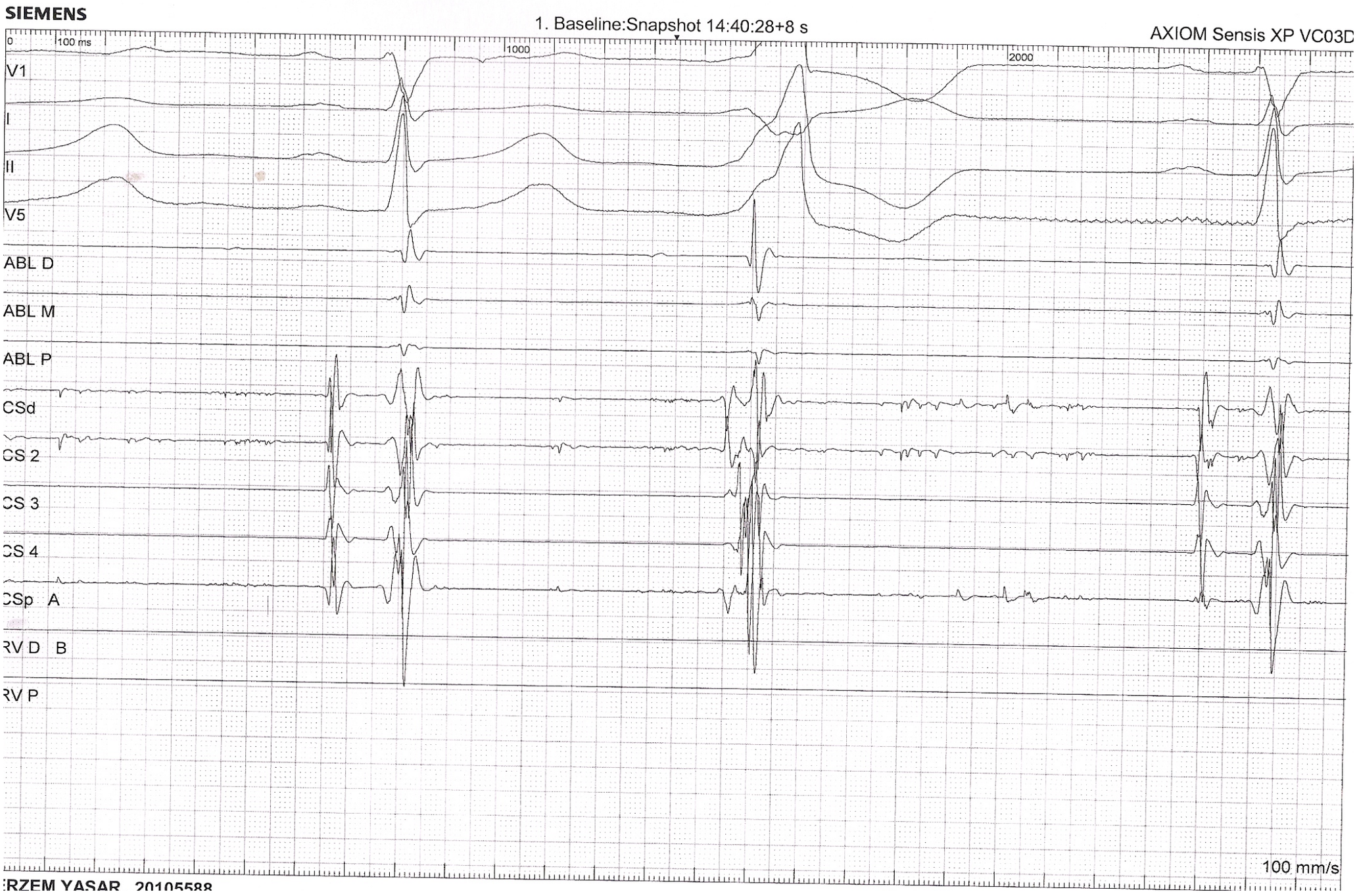
0
100 ms
1000
2000
I 10 mm/mV
II 10 mm/mV
III 10 mm/mV
aVL 10 mm/mV
aVF 10 mm/mV
-aVR 10 mm/mV
V1 10 mm/mV
V2 10 mm/mV
V3 10 mm/mV
V4 10 mm/mV
V5 10 mm/mV
V6 10 mm/mV
82
26
108
100 mm/s

Koroner Sinüs İçi Haritalama

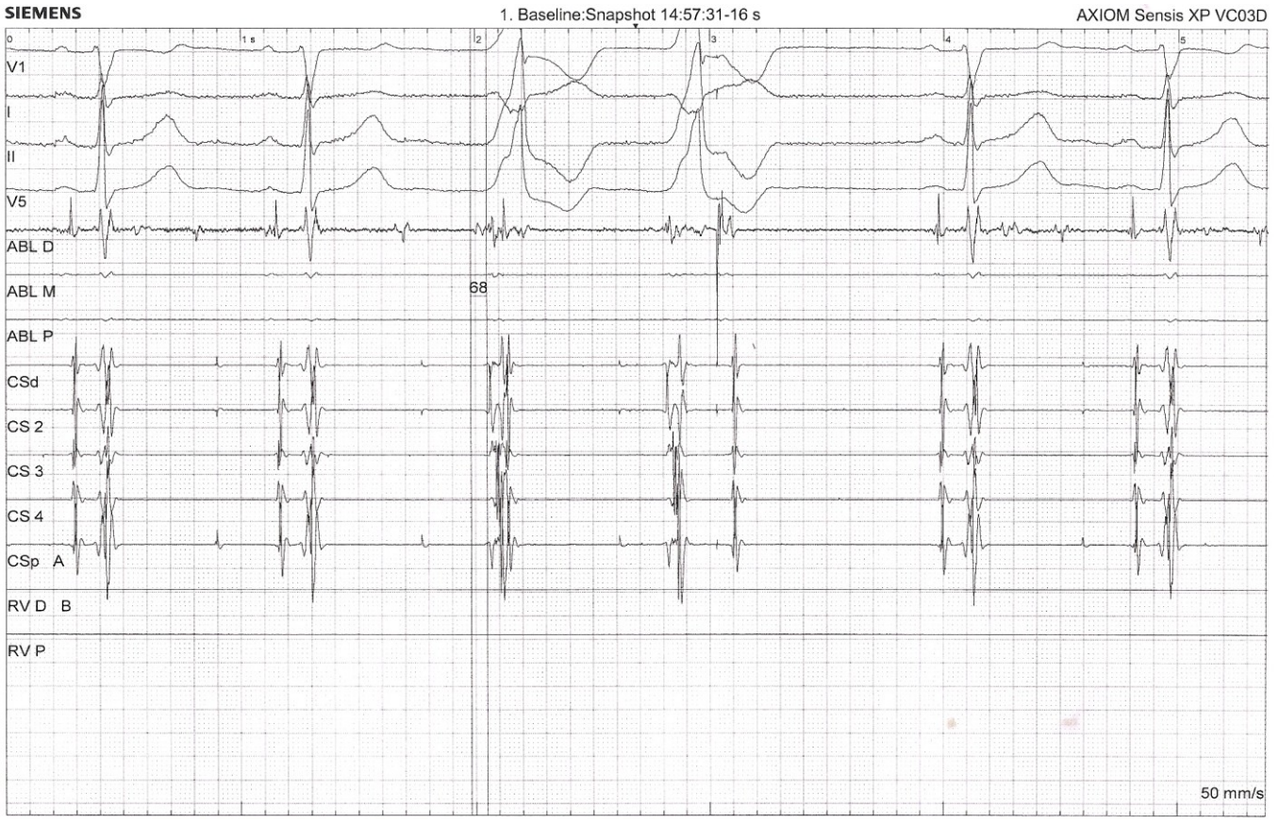


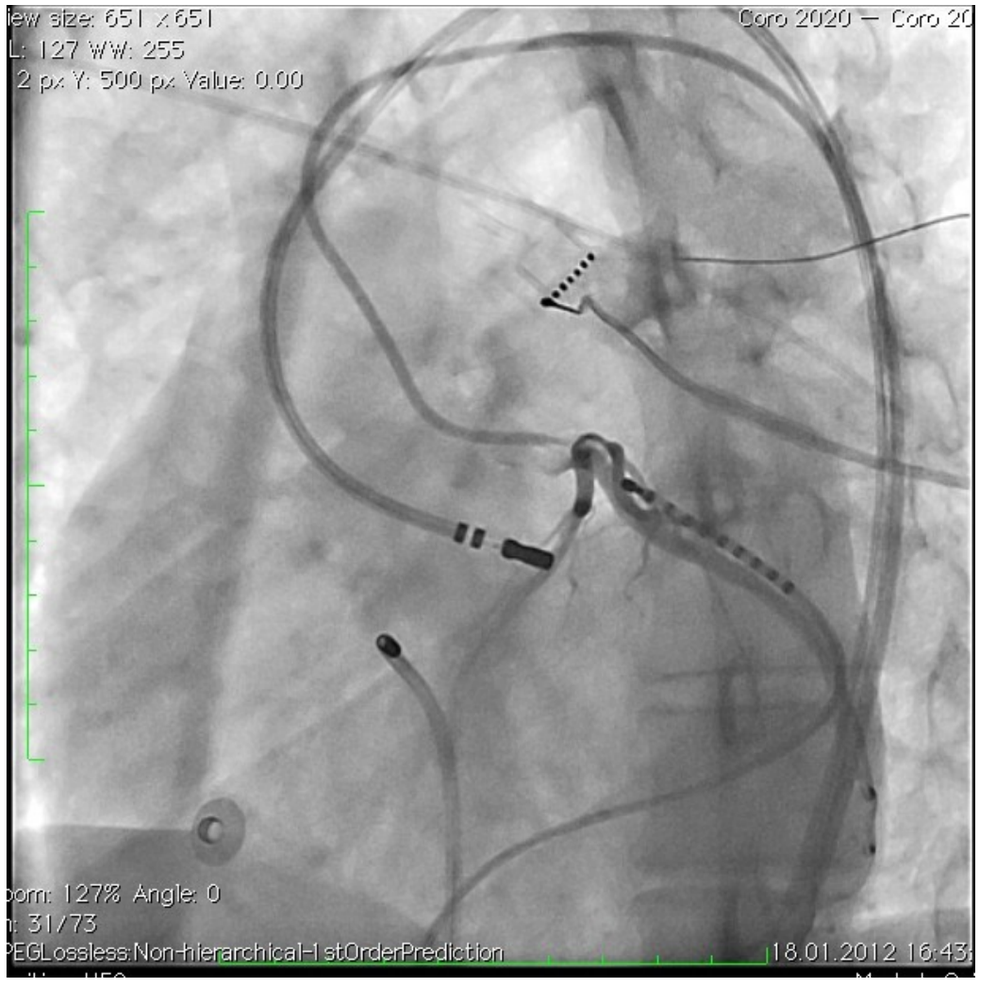


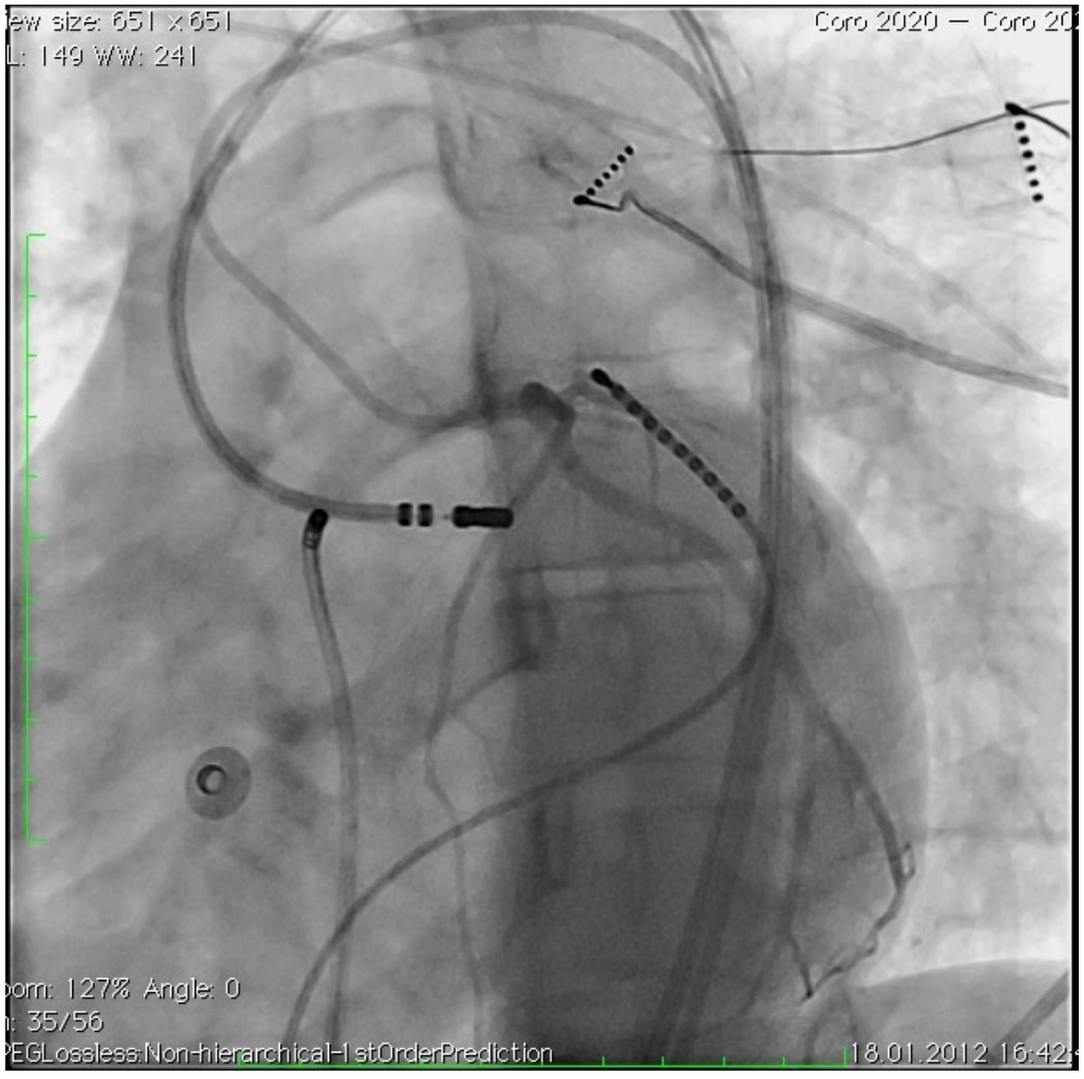
RVOT Haritalaması



Aort kusp haritalaması



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L: 149 WW: 241
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om: 127% Angle: 0
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EGLossless:Non-hierarchical-1stOrderPrediction
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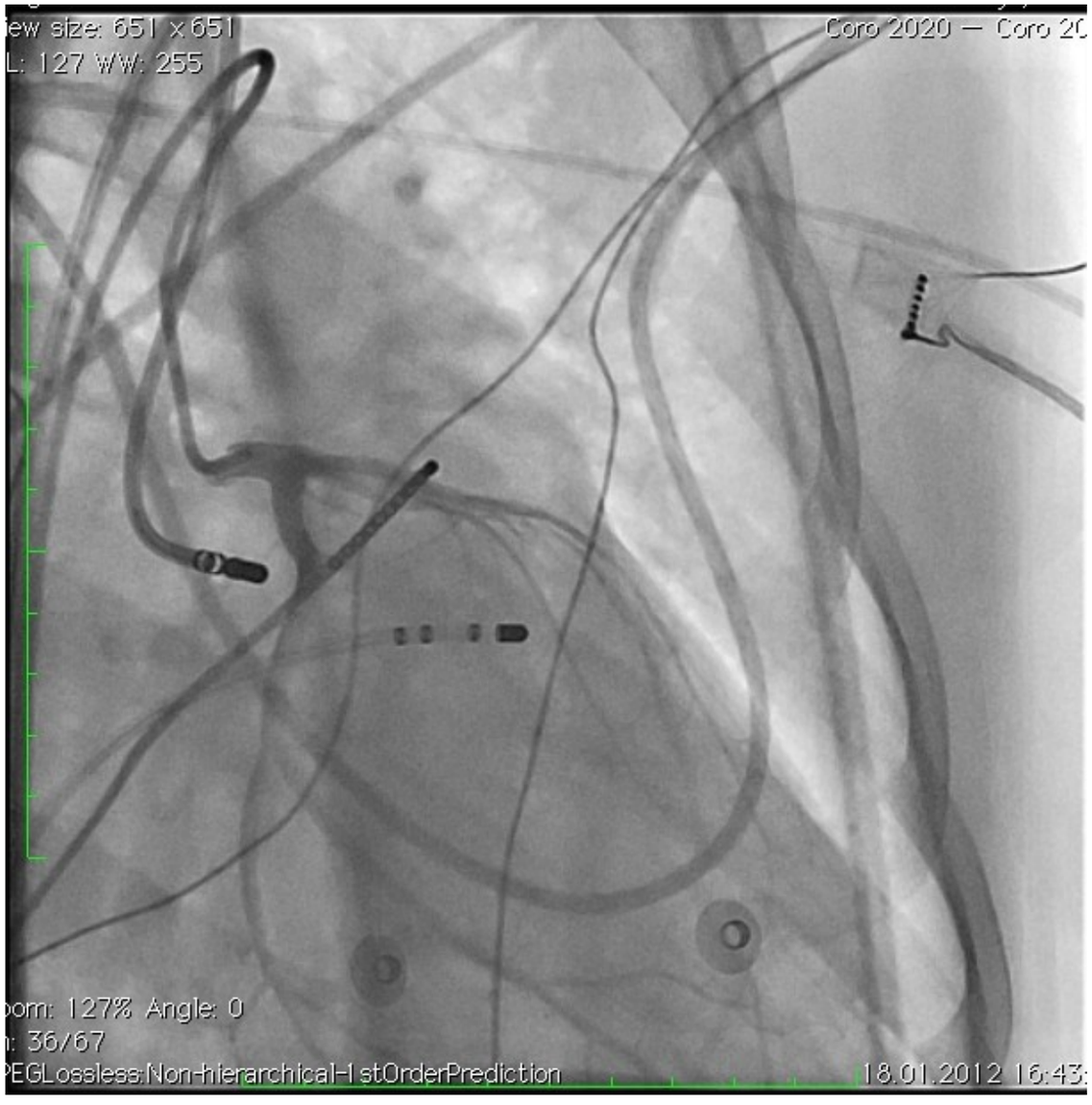
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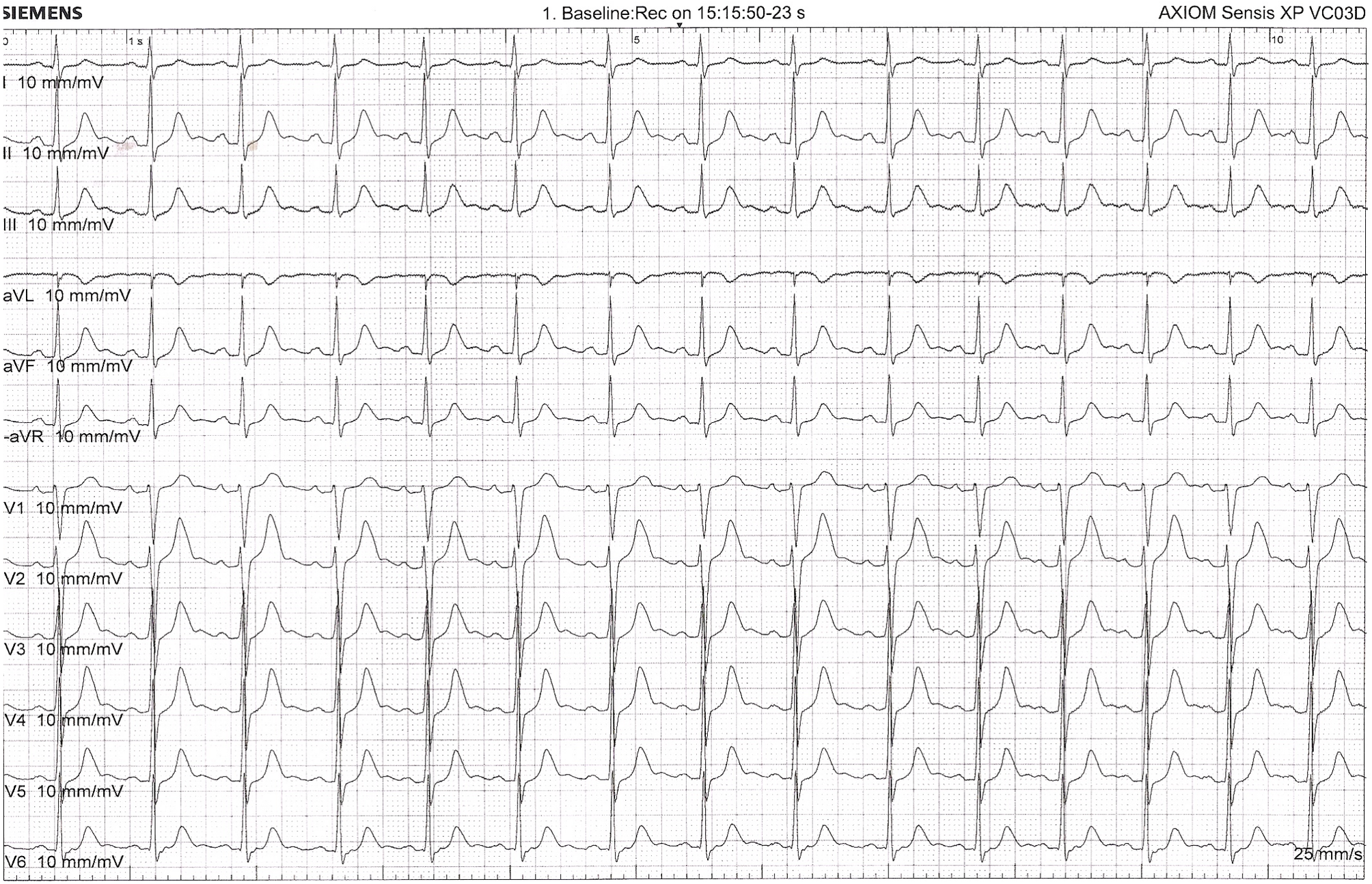
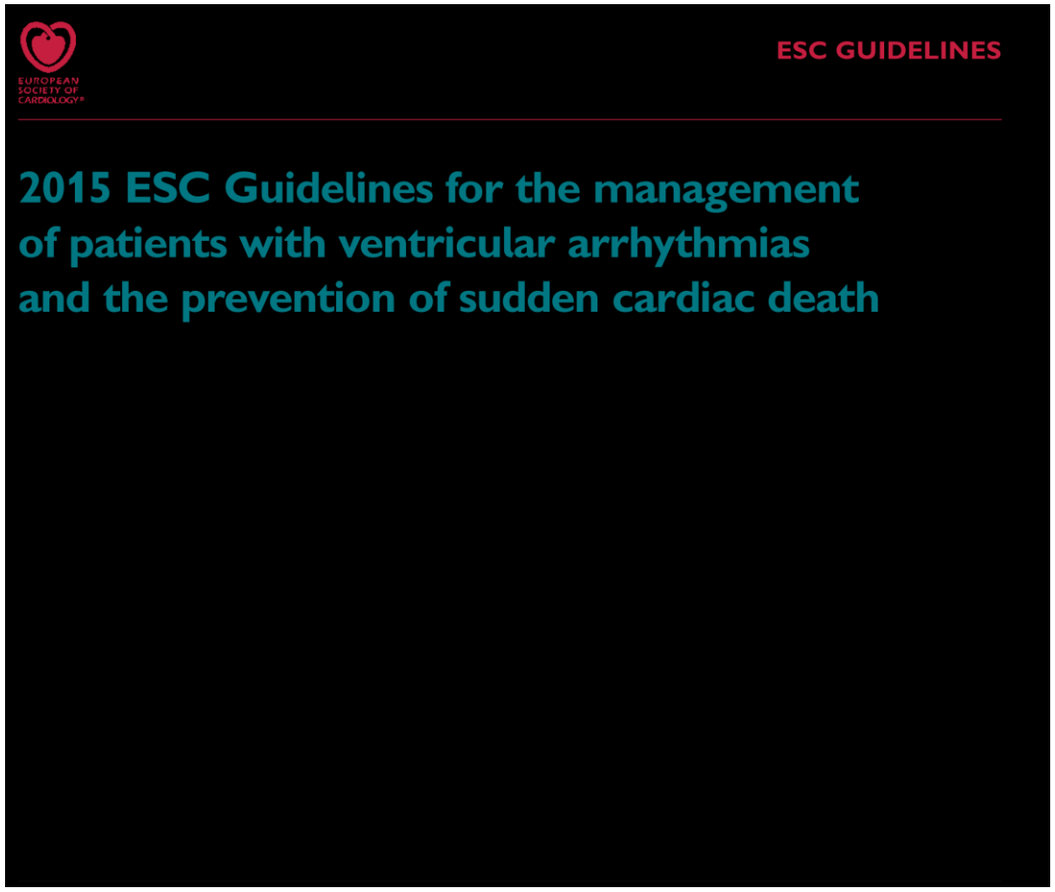
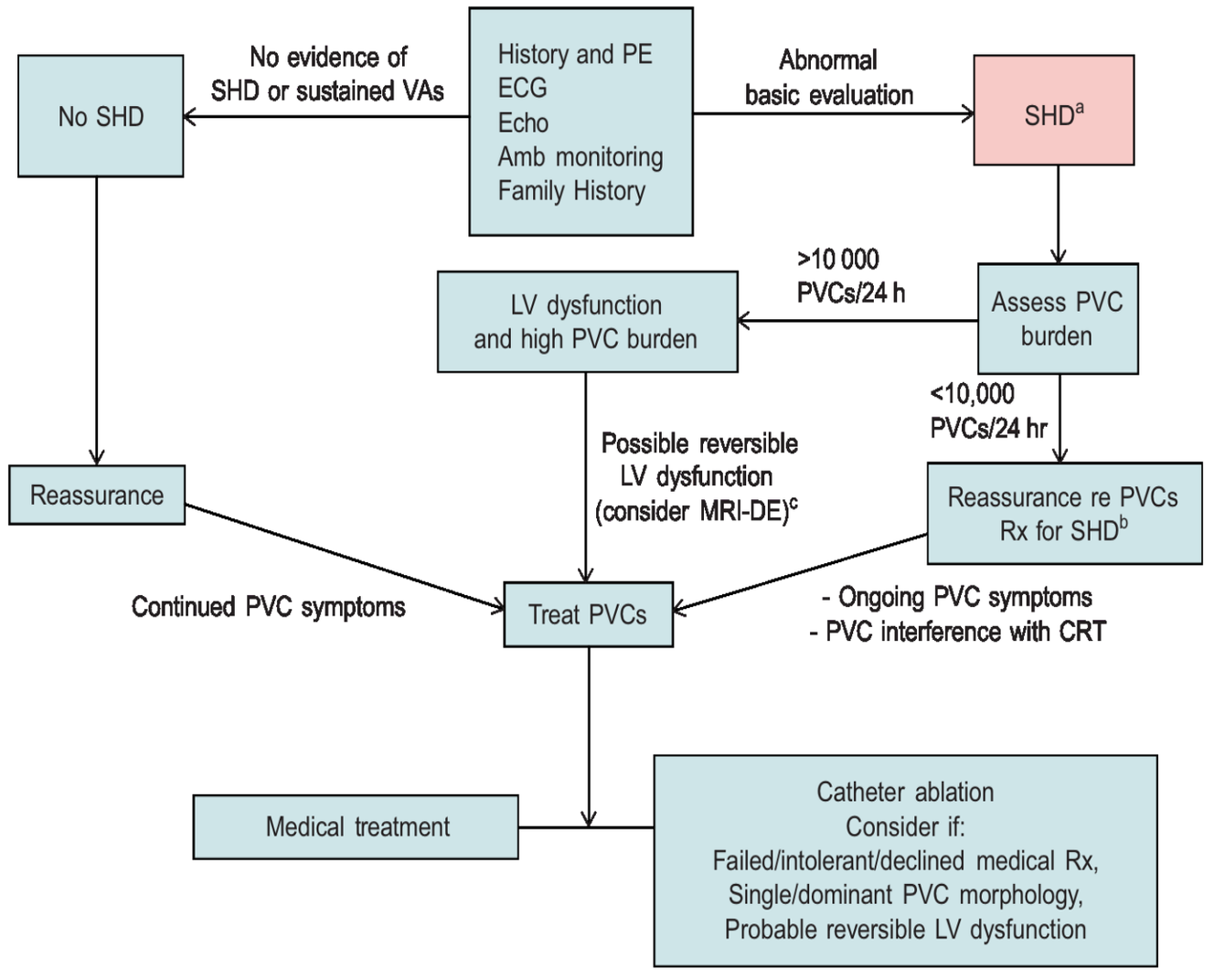
Coro 2020 — Coro 20
om: 127% Angle: 0
: 36/67
EGLossless:Non-hierarchical-1stOrderPrediction
18.01.2012 16:43:

Ablasyon sonu EKG



Prematüre Ventriküler Vurularda Tedavi





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Recommendations	Class[a]	Level[b]	Ref.[c]
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VEA’ların tetiklediği kardiyomiyopati

- First Evidence of Premature Ventricular Complex-Induced Cardiomyopathy: A Potentially Reversible Case of Heart Failure, Chugh SS, et al. JCE 2000;11:328-329
- 22 y, kadın hasta
- Şikayet:Çarpıntı ve halsizlik
- Holter: 25000-56000 PVCs, LBBB/inferior aks
- Ekokardiyografi:

		Ablasyondan 6 ay sonra	
• EF	%43----------	EF	%58
• LVEDD	65 mm----------	LVEDD	57 mm

VEA yükü ve Sol Ventrikül Disfonksiyonu

Table 1 PVC burden associated with LV dysfunction

	n	%LVd	%VEs LVd	%VEs normal LV	P	Predictive PVC burden
Ban et al.[21]	127 (28 LVd)	22%	31 + 11%	22 + 10%	0.001	26%
Deyell et al.[25]	90 (24 LVd)	27%	32 + 12%	27 + 12%	0.077	–
Munoz et al.[26]	70 (LVd 17)	24%	29 + 15%	17 + 14%	0.004	10% RV; 20% LV
Olgun et al.[27]	51 (21 LVd)	41%	30 + 11%	14 + 15%	0.0001	–
Hasdemir et al.[28]	249 (17 LVd)	7%	29 + 9%	8 + 7%	0.001	16%
Baman et al.[29]	174 (57 LVd)	33%	33 + 13%	13 + 12%	0.0001	24%
Kanei et al.[30]	108 (21 LVd)	19%	13 + 11%[a]	7 + 9%[a]	0.004	–

Lowest PVC count associated with LV dysfunction was 10% (Baman).

LV = left ventricle; LVD = left ventricular dysfunction; PVC = premature ventricular complexes; VE = ventricular ectopic.

[a]Assuming 100 000 beats/24 h.

Relationship between burden of premature ventricular complexes and left ventricular function

Timir S. Baman, MD,* Dave C. Lange, MD,* Karl J. Ilg, MD,* Sanjaya K. Gupta, MD,* Tzu-Yu Liu, MS,† Craig Alguire, MD,* William Armstrong, MD, FACC,* Eric Good, DO, FACC,* Aman Chugh, MD, FACC,* Krit Jongnarangsin, MD,* Frank Pelosi, Jr., MD,* Thomas Crawford, MD,* Matthew Ebinger, MD, DO,* Hakan Oral, MD, FACC,* Fred Morady, MD, FACC,* Frank Bogun, MD, FACC*

*From the *Division of Cardiovascular Medicine and the*
†Department of Electrical Engineering and Computer Science, University of Michigan, Ann Arbor, Michigan.

BACKGROUND Frequent idiopathic premature ventricular complexes (PVCs) can result in a reversible form of left ventricular dysfunction. The factors resulting in impaired left ventricular function are unclear. Whether a critical burden of PVCs can result in cardiomyopathy has not been determined.

OBJECTIVE The objective of this study was to determine a cutoff PVC burden that can result in PVC-induced cardiomyopathy.

METHODS In a consecutive group of 174 patients referred for ablation of frequent idiopathic PVCs, the PVC burden was determined by 24-hour Holter monitoring, and transthoracic echocardiograms were used to assess left ventricular function. Receiver-operator characteristic curves were constructed based on the PVC burden and on the presence or absence of reversible left ventricular dysfunction to determine a cutoff PVC burden that is associated with left ventricular dysfunction.

RESULTS A reduced left ventricular ejection fraction (mean 0.37 $\pm$ 0.10) was present in 57 of 174 patients (33%). Patients with a decreased ejection fraction had a mean PVC burden of 33% $\pm$ 13% as compared with those with normal left ventricular function 13% $\pm$ 12% (P <.0001). A PVC burden of >24% best separated the patient population with impaired as compared with preserved left ventricular function (sensitivity 79%, specificity 78%, area under curve 0.89) The lowest PVC burden resulting in a reversible cardiomyopathy was 10%. In multivariate analysis, PVC burden (hazard ratio 1.12, 95% confidence interval 1.08 to 1.16; P <.01) was independently associated with PVC-induced cardiomyopathy.

CONCLUSION A PVC burden of >24% was independently associated with PVC-induced cardiomyopathy.

KEYWORDS Premature ventricular complexes, Ablation, Cardiomyopathy

ABBREVIATIONS **CI** = confidence interval; **EF** = ejection fraction; **HR** = hazard ratio; **LV** = left ventricular; **PVC** = premature ventricular complexes; **ROC** = receiver operator characteristic; **RVOT** = right ventricular outflow tract

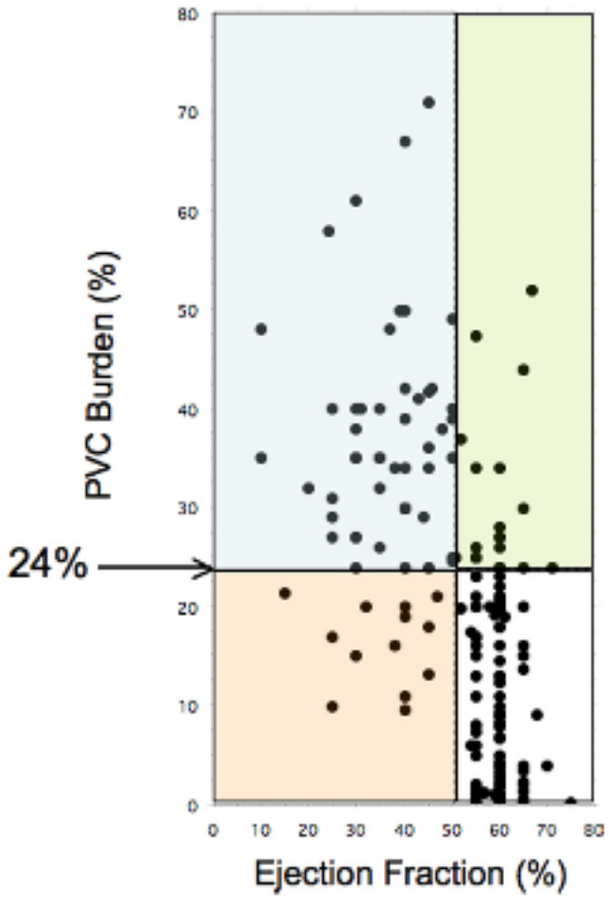
(Heart Rhythm 2010;7:865–869)

Table 1 Comparison of patients with frequent PVCs with and without cardiomyopathy

Clinical characteristics	No cardiomyopathy (n = 117)	Cardiomyopathy (n = 57)	*P* value
Male sex	52 (44)	35 (61)	.02
Age, years	48 ± 12	49 ± 12	.73
Mean duration of palpitations, months	57 ± 89	62 ± 100	.76
EF, %	59 ± 4	35 ± 9	<.01
Baseline LV end-diastolic dimension, mm	51 ± 6	57 ± 6	<.01
Baseline LV end-systolic dimension, mm	33 ± 7	42 ± 8	<.01
Therapy			
Beta-blockers/calcium channel blockers	90 (77)	44 (77)	.50
Angiotensin-converting enzyme inhibitors/angiotensin receptor blockers	23 (20)	27 (47)	<.01
Digoxin	2 (2)	4 (7)	.06
Antiarrhythmic drug therapy including amiodarone	29 (25)	12 (21)	.83
Amiodarone	4 (3)	5 (9)	.09

Values are n (%) or mean ± SD.

EF = ejection fraction; LV = left ventricular; PVC = premature ventricular complex.



- VEA yükü %24 ve üzeri olması , korunmuş LV sistolik fonksiyonlu hastaları bozulmuş LV sistolik fonksiyonu olan hastalardan ayırmada en iyisi (Sensitivite %79, spesifisite %78)

Table 1 Comparison of patients with frequent PVC with and without cardiomyopathy

Clinical characteristics	Cardiomyopathy (n = 76)	No cardiomyopathy (n = 165)	*P* value
Male, n (%)	51 (67)	64 (39)	<.0001
Age (y), mean ± SD	48 ± 16	48 ± 13	.98
Hypertension	23 (30)	38 (23)	.23
Left ventricular ejection fraction, mean ± SD	0.36 ± 0.09	0.59 ± 0.05	<.0001
PVC burden (%), mean ± SD	28 ± 12	15 ± 13	<.0001
Duration of palpitations (mo),* mean ± SD	135 ± 118	35 ± 52	<.0001
Asymptomatic status, n (%)	36 (47)	25 (15)	<.0001
Therapy, n (%)			
Beta blockers	57 (75)	116 (70)	.45
Calcium-channel blockers	16 (21)	35 (21)	.98
Angiotensin-converting enzyme inhibitors/angiotensin receptor blockers	33 (43)	23 (14)	<.0001
Digoxin	2 (3)	3 (2)	.65
Antiarrhythmic drug therapy including amiodarone	9 (12)	34 (21)	.99

PVC, premature ventricular complex.

*Duration in symptomatic patients.

VEA kökenli kardiyomiyopati

- Net olarak tanımlanmış klinik bir durum, ancak hala
- Sık VEA olan hastaların çoğunluğu benign bir seyir göstermekte, bununla beraber hastaların 3'te birine kadar kısmında KMP gelişebilmekte.
- Bu yüzden, VEA-KMP'yi çok yoğun VEA olan ve açıklanamayan kardiyomiyopati hastalarında düşünmek gerekir.
- Kateter ablasyon yöntemi, sık VEA olan semptomatik ve/veya kalp yetmezliği bulguları olan hastalar için faydası ortaya konmuş bir tedavi yöntemidir.
- SORU: Sık VEA'ları tedavi etmek için ne kadar beklemeliyiz?

Relation of symptoms and symptom duration to premature ventricular complex–induced cardiomyopathy

Miki Yokokawa, MD, Hyungjin Myra Kim, ScD, Eric Good, DO, Aman Chugh, MD, Frank Pelosi, Jr, MD, Craig Alguire, MD, William Armstrong, MD, Thomas Crawford, MD, Krit Jongnarangsin, MD, Hakan Oral, MD, Fred Morady, MD, Frank Bogun, MD

From the Division of Cardiovascular Medicine, University of Michigan, Ann Arbor, Michigan.

BACKGROUND Frequent idiopathic premature ventricular complexes (PVCs) can result in a reversible form of cardiomyopathy. In this study, the determinants of PVC-induced left ventricular (LV) dysfunction were assessed.

METHODS The subjects of this study were 241 consecutive patients (115 men [48%], mean age 48 ± 14 years) referred for ablation of frequent PVCs. One hundred eighty patients (75%) experienced palpitations and 61 (25%) did not. The PVC burden was determined by 24-hour Holter monitoring, and echocardiograms were performed to assess LV function. An LV ejection fraction of <50% was considered abnormal.

RESULTS LV ejection fraction (mean 0.36 ± 0.09) was present in 76 of 241 patients (32%). There was a higher prevalence of males among the patients with PVC cardiomyopathy compared to patients with normal LV function (51/76 [67%] vs 64/165 [39%]; $P < .0001$). The mean PVC burden was significantly higher in patients with PVC cardiomyopathy than in patients with normal LV function (28% ± 12% vs 15% ± 13%; $P < .0001$). Among symptomatic patients, those with cardiomyopathy had a significantly longer duration of palpitations (135 ± 118 months) compared with patients with normal LV function (35 ± 52 months; $P < .0001$). The proportion of asymptomatic patients was significantly higher in the presence of cardiomyopathy (36/76, 47%) than in normal LV function (25/165, 15%; $P < .0001$). Symptom duration of 30 to 60 months, symptom duration >60 months, the absence of symptoms, and the PVC burden in asymptomatic patients were independent predictors of impaired LV function (adjusted odds ratio [95% confidence interval]: 4.0 [1.1–14.4], 20.1 [6.3–64.1], 13.1 [4.1–37.8], and 2.1 [1.2–3.6], respectively).

CONCLUSIONS The duration of palpitations and the absence of symptoms are independently associated with PVC-induced cardiomyopathy.

KEYWORDS Premature ventricular complexes; Ablation; Cardiomyopathy

ABBREVIATIONS **LV** = left ventricular; **PVCs** = premature ventricular complexes

(Heart Rhythm 2012;9:92–95)

- Sık VEA'lerin ablasyonu için refere edilen 241 ardışık hastanın retrospektif analizi (115 erkek, ortalama yaş 48±14 yıl)
- 180 hasta (%75) çarpıntı
- 61 hasta (%25), çarpıntı şikayeti yok.
- VEA yoğunluğu ve LV fonksiyonların analizi (Ekokardiyografik)

Sık VEA, sistolik fonksiyonlar ve semptomlar

Clinical characteristics	Cardiomyopathy (n = 76)	No cardiomyopathy (n = 165)	*P* value
Male, n (%)	51 (67)	64 (39)	<.0001
Age (y), mean ± SD	48 ± 16	48 ± 13	.98
Hypertension	23 (30)	38 (23)	.23
Left ventricular ejection fraction, mean ± SD	0.36 ± 0.09	0.59 ± 0.05	<.0001
PVC burden (%), mean ± SD	28 ± 12	15 ± 13	<.0001
Duration of palpitations (mo), *mean ± SD	135 ± 118	35 ± 52	<.0001
Asymptomatic status, n (%)	36 (47)	25 (15)	<.0001
Therapy, n (%)			
Beta blockers	57 (75)	116 (70)	.45
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PVC, premature ventricular complex.
∗ Duration in symptomatic patients.

Sık VEA, sistolik fonksiyonlar ve semptomlar

Table 2 Predictors of PVC cardiomyopathy based on multiple logistic regression model

Clinical characteristics	Adjusted odds ratio	95% confidence interval	*P* value
Asymptomatic status	13.1	4.1–37.8	<.001
Duration of palpitations (mo)			
<30	1.0	—	—
30–60	4.0	1.1–14.4	.03
>60	20.1	6.3–64.1	<.001
PVC burden in asymptomatic patients*	2.1	1.2–3.6	.007

PVC, premature ventricular complex.

*PVC burden centered at the average value of 19% and divided by 10, and so a 1-unit increase corresponds to a 10% increase in PVC burden.

[redacted]

(J Cardiovasc Electrophysiol, Vol. 22, pp. 791-798, July 2011)

TABLE 1
Patient Characteristics

	EF < 50% (n = 17)		EF > = 50% (n = 53		
	Mean or N	**SD or %**	**Mean or N**	**SD or %**	**P value**
Age (years)	42.5	16.6	39.1	17.7	0.495
Males	10	58.8	20	37.7	0.126
Duration of symptoms (months)	45.6	64.2	47.4	62.9	0.927
NYHA dyspnea class	1.17	0.58	1.06	0.24	0.548
Palpitations	10	58.8	48	94.1	**0.0004**
Dizziness	6	35.3	32	62.8	**0.048**
Fatigue	2	13.3	9	22.5	0.449
Coronary artery disease	2	11.8	3	5.7	0.395
Hypertension	1	5.9	12	22.6	0.122
Antiarrhythmics	6	30	15	28.9	0.148
B-blockers	15	88.2	35	66	0.078
Ca-blockers	2	11.8	21	39.6	0.101
ACE-I	5	50	6	20	0.066
ARB	1	10	1	3.3	0.402
LVEF (%)	38.5	8.8	58.8	6.1	**<0.0001**
LVEDd (mm)	59.4	7.1	51.2	5.2	**0.0002**
Number PVCs/24 hours	29,906	16,304	17,951	15,870	**0.01**
PVC burden (%)	29.3	14.6	16.7	13.7	**0.004**
Nonsustained VT	13	76	21	40	**0.001**

NYHA = New York Heart Association; ACE-I = angiotensin converting enzyme inhibitor; ARB = angiotensin receptor blocker; LVEF = left ventricular ejection fraction; LVEDd = left ventricular end-diastolic diameter. Bold values refer to the reported symptoms of dizziness and/or palpitations with regard to reduced LVEF.

Beneficial effects of catheter ablation on left ventricular and right ventricular function in patients with frequent premature ventricular contractions and preserved ejection fraction

Adrianus P Wijnmaalen, Victoria Delgado, Martin J Schalij,
Carine F B van Huls van Taxis, Eduard R Holman, Jeroen J Bax, Katja Zeppenfeld

Heart 2010;**96**:1275–1280. doi:10.1136/hrt.2009.188722

	Kontrol (n:25)	RVME (n:49)	P value
LVEF(%)	58±5	56±7	0.201
LV radial strain (%)	43.3±12.7	30.5±12.9	<0.001
LV circumferential strain (%)	-20.2±3.1	-15.8±4.0	<0.001
LV longitudinal strain (%)	-20.1±1.0	-17.4±3.0	<0.001
RV longitudinal strain (%)	-30.1±4.4	-23.8±6.7	<0.001

Doğal Seyir?

- 239 hasta

  >1000 VEA/24 saat, LBBB/inferior aks

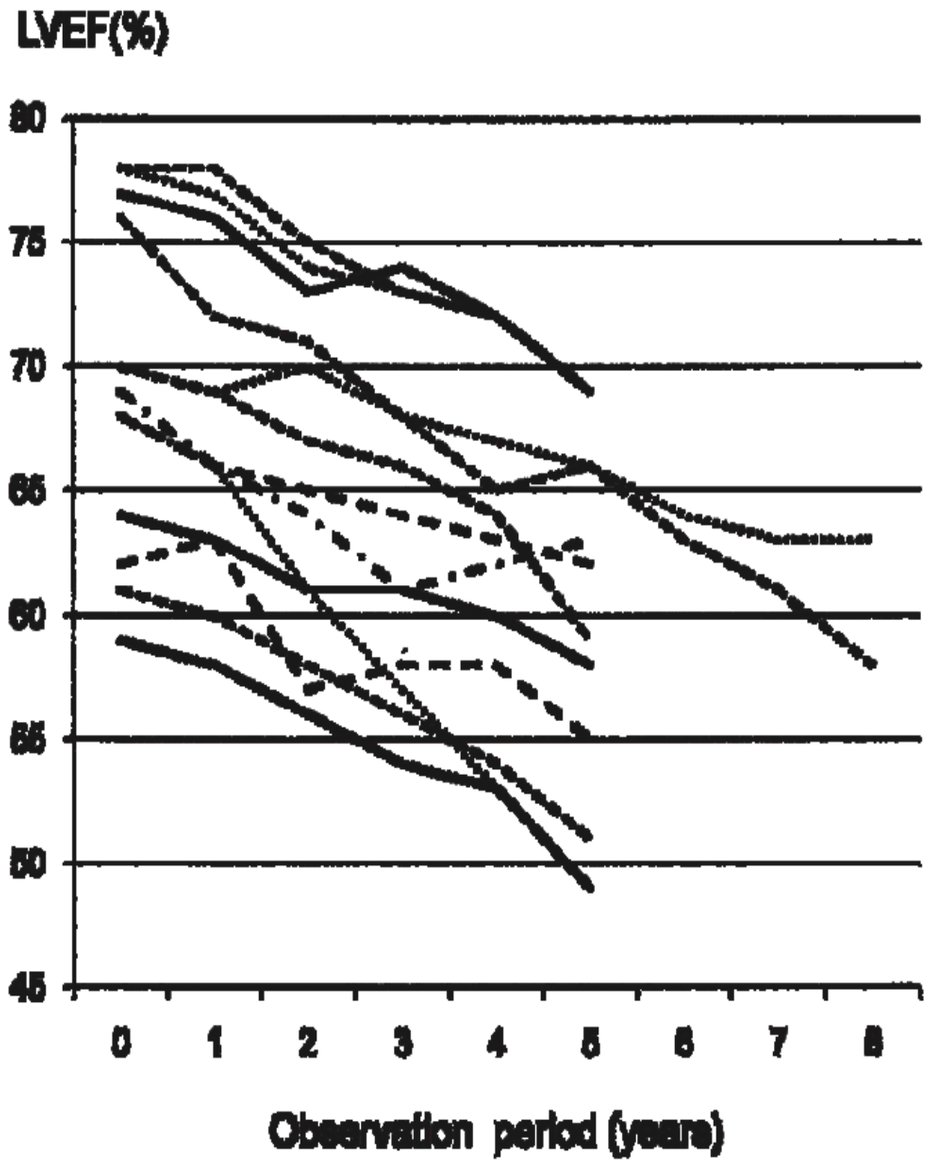
  . >20000 VEA (n:46)

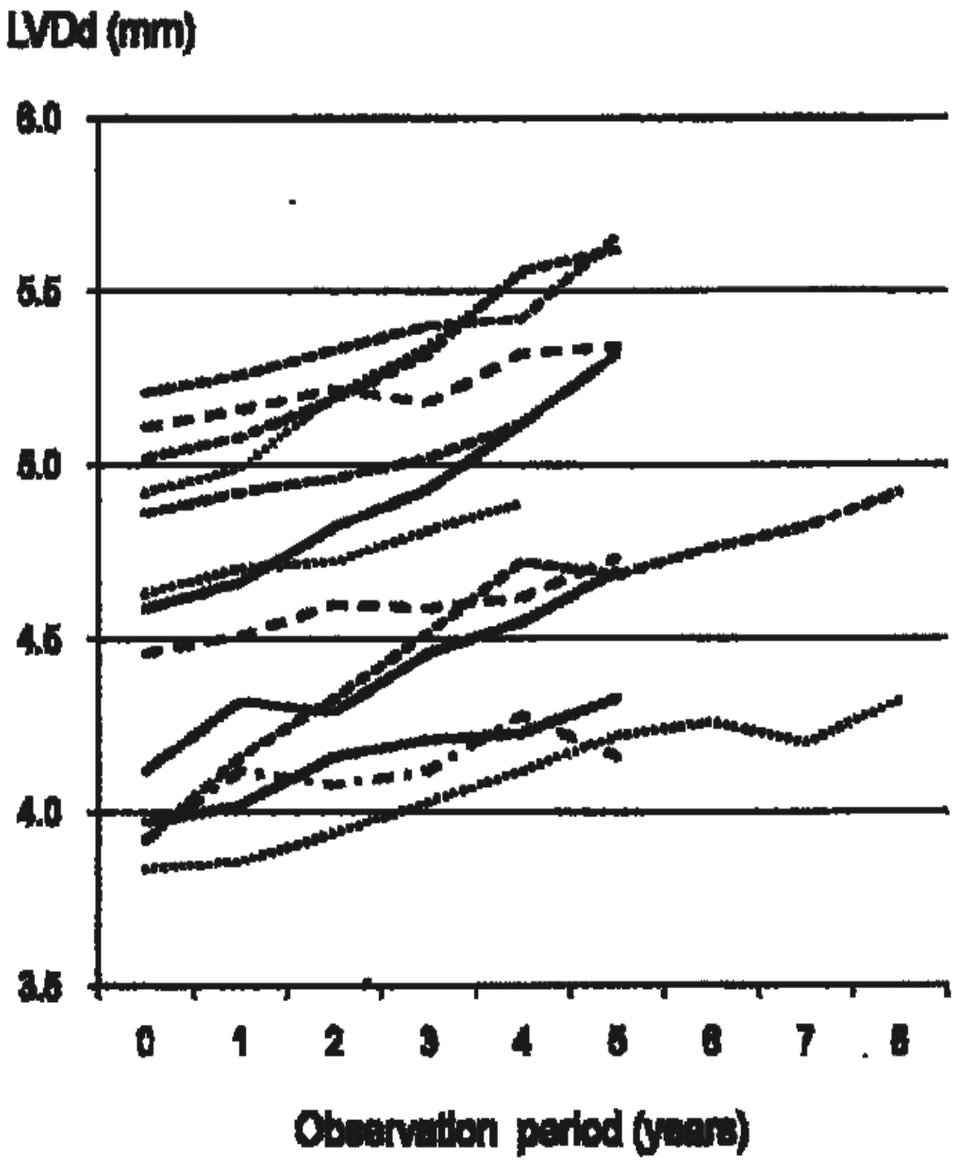
  . >5000-20000 VEA (n:105)

  . >1000-5000 VEA (n:88)
- Asemptomatik, normal LV fonksiyonları
- İzlem süresi:4 yıl (189/239 hastada ilave olarak 1-4 yıl arasında fazladan takip edilmiş)

Niwano S et al Heart 2009;95:1230-1237

Doğal Seyir


LVEF(%)
80
75
70
65
60
55
50
45
0
1
2
3
4
5
6
7
8
Observation period (years)


LVDd (mm)
6.0
5.5
5.0
4.5
4.0
3.5
0
1
2
3
4
5
6
7
8
Observation period (years)

Beneficial effects of catheter ablation of frequent premature ventricular complexes on left ventricular function

Minhua Zang, Tuo Zhang, Jialiang Mao, Shengheng Zhou, Ben He

Table 1 Design and selection criteria of included studies

Source	Year	Study design	Inclusion/exclusion criteria
Sekiguchi	2005	Retrospective	Patients with severe symptoms related to monomorphic frequent PVCs without evidence of structural heart disease. No unnecessary catheter ablation was ever performed.
Takemoto	2005	Retrospective	Patients with monomorphic frequent RVOT-PVC (>20% PVCs) without evidence of structural heart disease.
Lelakowski	2009	Prospective	Patients with symptomatic, persistent, monomorphic PVCs and LVEF >50% at baseline. No evidence of structural heart disease.
Sarrazin	2009	Retrospective	Patients with frequent PVCs with a remote myocardial infarction.
Kim subgroup	2010	Retrospective	Patients with symptomatic frequent PVCs (>4% PVCs) without evidence of structural heart disease. Patients of this study were from the same population in Ban's study, subgroup data of patients with normal LV function was included from this study.
Wijnmaalen	2010	Retrospective	Patients with symptomatic, frequent PVCs (>5% PVCs) and LVEF >50% at baseline. No evidence of structural heart disease.
Del Carpio Munoz	2011	Retrospective	Patients with symptomatic, monomorphic frequent PVCs without evidence of structural heart disease.
Mountantonakis	2011	Retrospective	Patients with frequent PVCs and LVEF <50% at baseline. No evidence of active or prior infarction.
Ban	2012	Retrospective	Patients with frequent PVCs (>10% PVCs) and LVEF <50% at baseline. No evidence of structural heart disease.
Kuroki	2012	Retrospective	Patients with frequent PVCs without evidence of structural heart disease.
Lakkireddy	2012	Prospective	Patients were considered non-responders to CRT with high PVC burden.
Lu	2012	Retrospective	Patients with frequent PVCs and LVEF <50% at baseline.
Yokokawa	2012	Retrospective	Patients with frequent PVCs without evidence of structural heart disease.
Yokokawa subgroup	2013	Retrospective	Patients with frequent PVCs and LVEF <50% at baseline. Data of this study were only included in subgroup analysis.
Penela	2013	Prospective	Patients with LVEF <50% of any aetiology and frequent and/or symptomatic PVCs (>4% PVCs). No patient was excluded because of the number of PVC morphologies or the presumed site of origin based on electrocardiography.

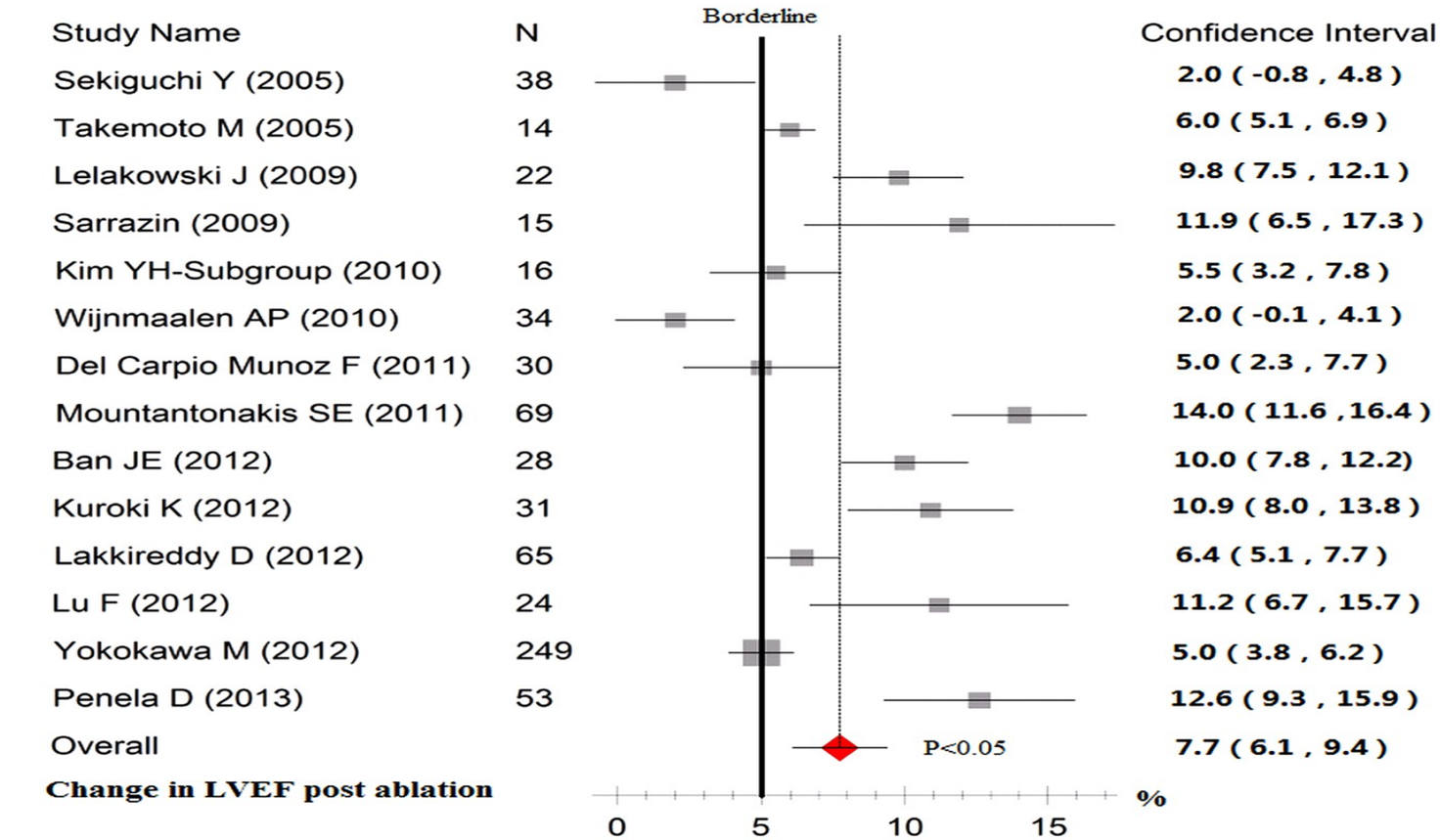
Data reported as mean±SD or median (IQR) as original manuscript.
CRT, cardiac resynchronisation therapy; NA, no data available; PVCs, premature ventricular complexes; RVOT, RV outflow tract.

Table 2 General characteristics of included studies

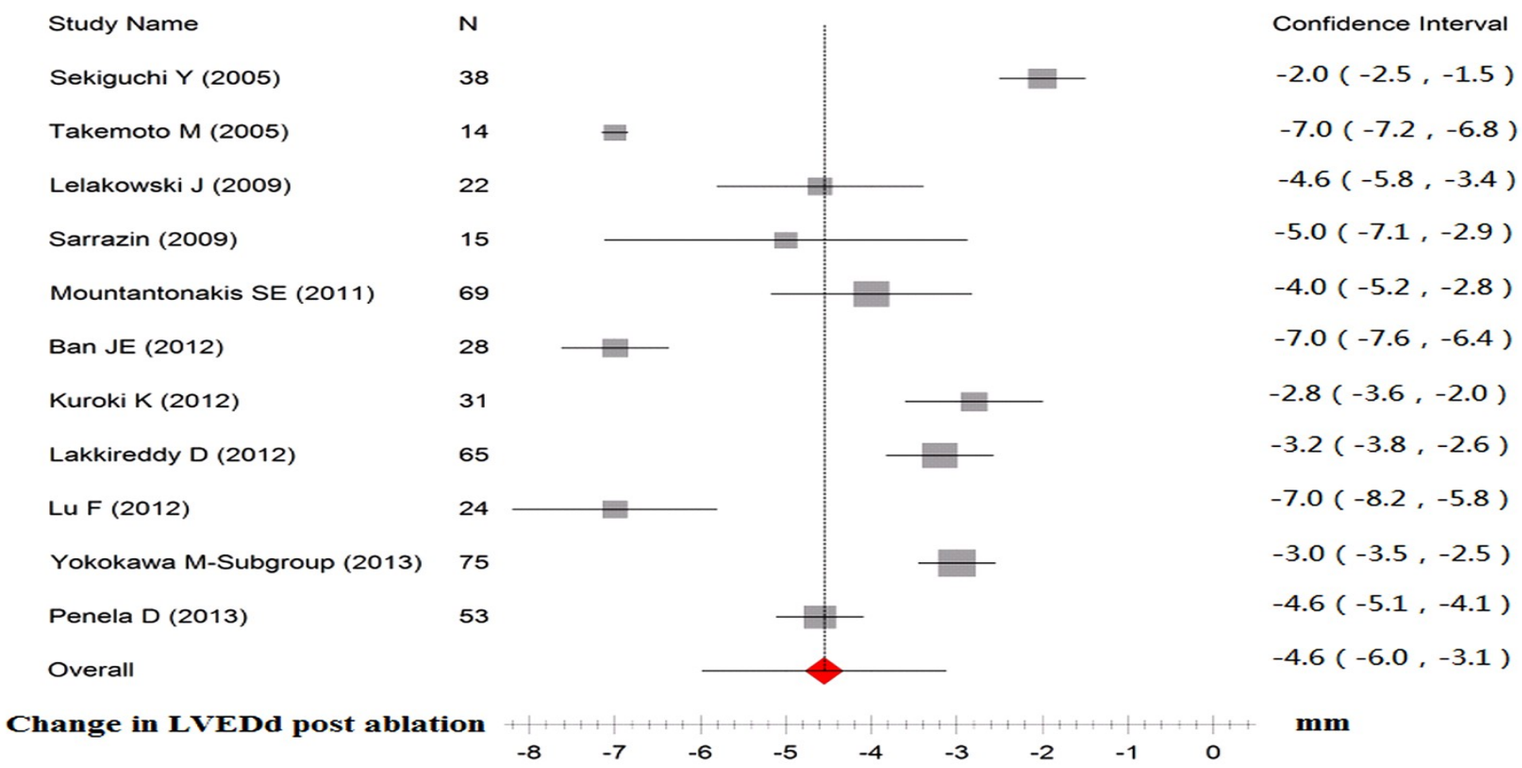
Source	Year	Country	Period of recruitment	Total no. of patients	Mean age (year)	SOO of PVCs		LVEF at baseline (%)	PVCs burden at baseline (%)	Procedure complication (%)	Echocardiography follow-up (month)	Long-term success rate (%)
						RV (%)	OT (%)					
Sekiguchi	2005	Tokyo, Japan	2003–2004	38	58±13	60	97	65±11	NA	0%	8±2	81
Takemoto	2005	Kitakyushu, Japan	1994–2004	14	52±4	100	100	66±2	34±3	3% (femoral arteriovenous fistula)	8±1	90
Lelakowski	2009	Krakow, Poland	2004–2007	22	38±8	73	82	58±7	NA	NA	6	NA
Sarrazin	2009	Michigan, USA	NA	15	59±12	13	65	38±11	22±12	0%	2±1	NA
Kim subgroup*	2010	Seoul, Korea	2006–2009	16	NA	81	81	NA	NA	1% (tamponade)	10±7	90
Wijnmaalen	2010	Leiden, Netherlands	NA	34	49±16	71	76	56±7	26±13	0%	13 (5–22)	NA
Del Carpio Munoz	2011	Mayo, USA	2005–2008	30	40±17	61	64	54±11	20±15	NA	1–6	NA
Mountantonakis	2011	Pennsylvania, USA	NA	69	51±16	39	100	35±9	29±13	8% (haematoma, aneurysm)	11±6	88
Ban	2012	Seoul, Korea	NA	28	48±14	82	89	44±5	31±11	NA	4±3	83
Kuroki	2012	Ibaraki, Japan	2008–2010	31	52±18	52	65	56±10	NA	NA	7±1	NA
Lakkireddy	2012	USA, Canada, Italy	2007–2010	65	67±12	25	18	26±6	NA	3% (TIA, tamponade)	6	88
Lu	2012	Minnesota, USA	NA	24	60±15	29	46	32±15	15±6	0%	8	NA
Yokokawa	2012	Michigan, USA	NA	249	48±14	48	48	52±12	19±14	NA	3–4	NA
Yokokawa subgroup†	2013	Michigan, USA	NA	75	50±16	44	37	39±10	26±11	NA	NA	NA
Penela	2013	Spain, Netherlands, Argentina	2010–2012	53	53±12	45	60	34±13	22±13	5% (tamponade, haematoma, pericarditis, thromboembolism)	12	66

Data reported as mean±SD or median (IQR) as original manuscript.
*Subgroup data of patients with normal LV function were included from this study.
†Data of this study were only included in subgroup analysis.
OT, outflow tract; SOO, site of origin; TIA, transient ischaemic attack; other abbreviations as in table 1.

A


Borderline
Study Name
N
Confidence Interval
Sekiguchi Y (2005) 38 2.0 (-0.8 , 4.8)
Takemoto M (2005) 14 6.0 (5.1 , 6.9)
Lelakowski J (2009) 22 9.8 (7.5 , 12.1)
Sarrazin (2009) 15 11.9 (6.5 , 17.3)
Kim YH-Subgroup (2010) 16 5.5 (3.2 , 7.8)
Wijnmaalen AP (2010) 34 2.0 (-0.1 , 4.1)
Del Carpio Munoz F (2011) 30 5.0 (2.3 , 7.7)
Mountantonakis SE (2011) 69 14.0 (11.6 ,16.4)
Ban JE (2012) 28 10.0 (7.8 , 12.2)
Kuroki K (2012) 31 10.9 (8.0 , 13.8)
Lakkireddy D (2012) 65 6.4 (5.1 , 7.7)
Lu F (2012) 24 11.2 (6.7 , 15.7)
Yokokawa M (2012) 249 5.0 (3.8 , 6.2)
Penela D (2013) 53 12.6 (9.3 , 15.9)
Overall P<0.05 7.7 (6.1 , 9.4)
Change in LVEF post ablation
%
0
5
10
15

B


Study Name
N
Confidence Interval
Sekiguchi Y (2005) 38 -2.0 (-2.5 , -1.5)
Takemoto M (2005) 14 -7.0 (-7.2 , -6.8)
Lelakowski J (2009) 22 -4.6 (-5.8 , -3.4)
Sarrazin (2009) 15 -5.0 (-7.1 , -2.9)
Mountantonakis SE (2011) 69 -4.0 (-5.2 , -2.8)
Ban JE (2012) 28 -7.0 (-7.6 , -6.4)
Kuroki K (2012) 31 -2.8 (-3.6 , -2.0)
Lakkireddy D (2012) 65 -3.2 (-3.8 , -2.6)
Lu F (2012) 24 -7.0 (-8.2 , -5.8)
Yokokawa M-Subgroup (2013) 75 -3.0 (-3.5 , -2.5)
Penela D (2013) 53 -4.6 (-5.1 , -4.1)
Overall -4.6 (-6.0 , -3.1)
Change in LVEDd post ablation
mm
-8
-7
-6
-5
-4
-3
-2
-1
0

Sonuç

- Asemptomatik yüksek sıklıkta VEA'lar zamanla ciddi kalp yetmezliğine sebep olabilirler.
- Eğer ablasyon yapılmaz ise, bu hastalar kesinlikle uzun dönemde yakından kronik kalp hastalığı gelişimi yönünden takip edilmelidirler.
- Asemptomatik VEA olan hastalarda ayrıca yüksek hızlı VT gelişebilir (daha az olasılıkla AKÖ)
- VEA'ların kateter ablasyonu iyileştirici bir tedavi opsiyonudur. (Özellikle deneyimli merkezlerde yüksek başarı oranında ve düşük komplikasyon oranları ile)
- Yüksek VEA'ları olan asemptomatik hastalarda, dikkatli bir şeklde hastaları izlemek ile erken iyileştirici tedavi olan kateter ablasyon yöntemi arasında bireysel olarak hasta bazında tartışılması gerekir.

- Sabrınız için teşekkürler……..