Sudden Death Internet Symposium

Polymorphic ventricular tachycardia in patients with vasospastic angina

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Introduction

*Development of polymorphic ventricular tachycardia (PVT) and sudden cardiac death (SCD) is frequently observed in the conditions of prolonged QT intervals.

*In the settings of normal QT intervals, PVT and SCD are mostly seen in acute myocardial infarction and catecholaminergic PVT (CPVT).

*Vasospastic angina is another condition to develop PVT and SCD with normal QT interval, although its incidences are relatively low, but should be kept in mind for clinical practice.

Case presentation-1 ; 57 yrs, male

[Clinical symtom]; Syncope

[Past history]; Hypertension, AF, gout

[Family history]; np

[Present illness];

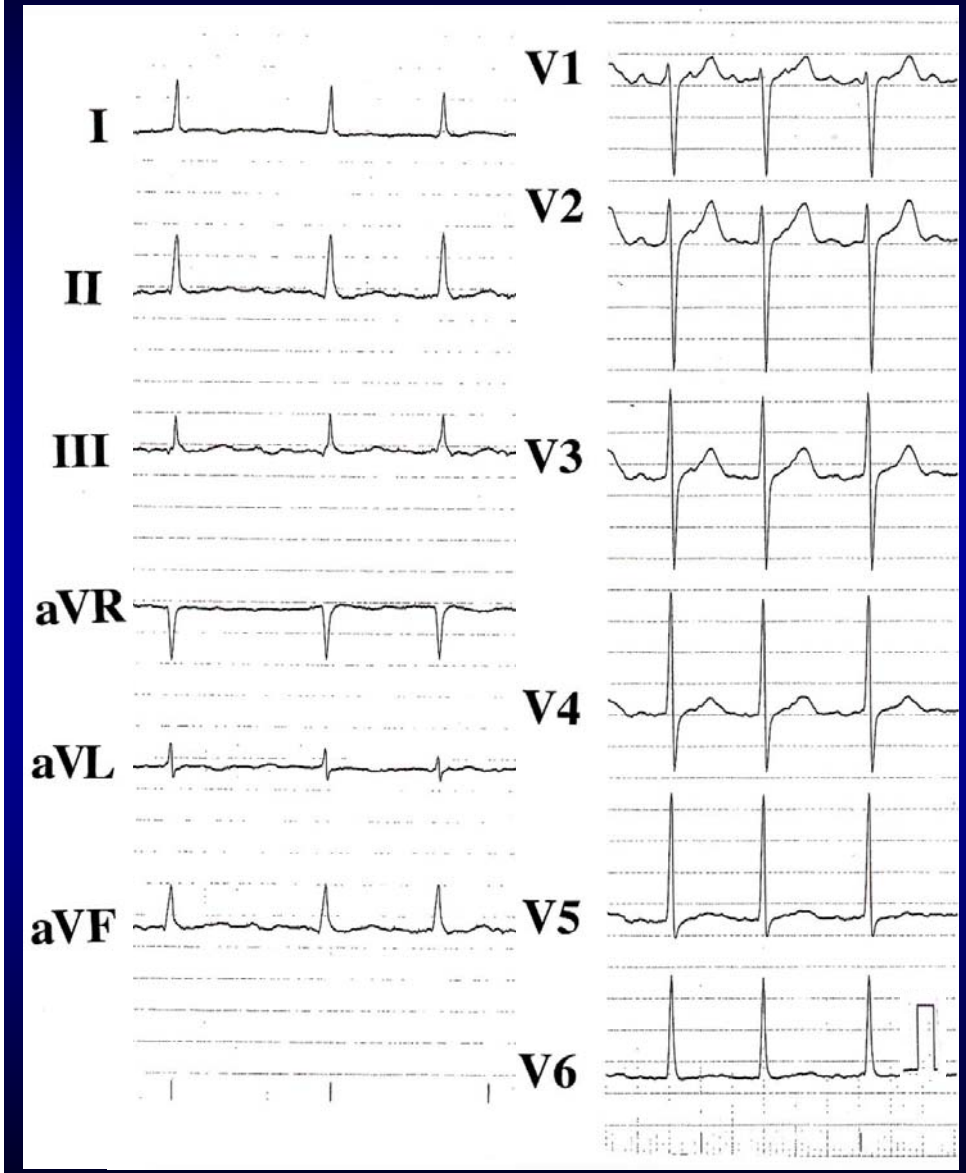
*** 03/2001** During admission to other hospital because of embolism in superior iliac artery, he experienced frequent episodes of chest pain. His ECG monitor demonstrated ST elevation and development of VT/VF associated with chest pain.

* Coronary angiogram demonstrated multiple coronary arterial spasms.

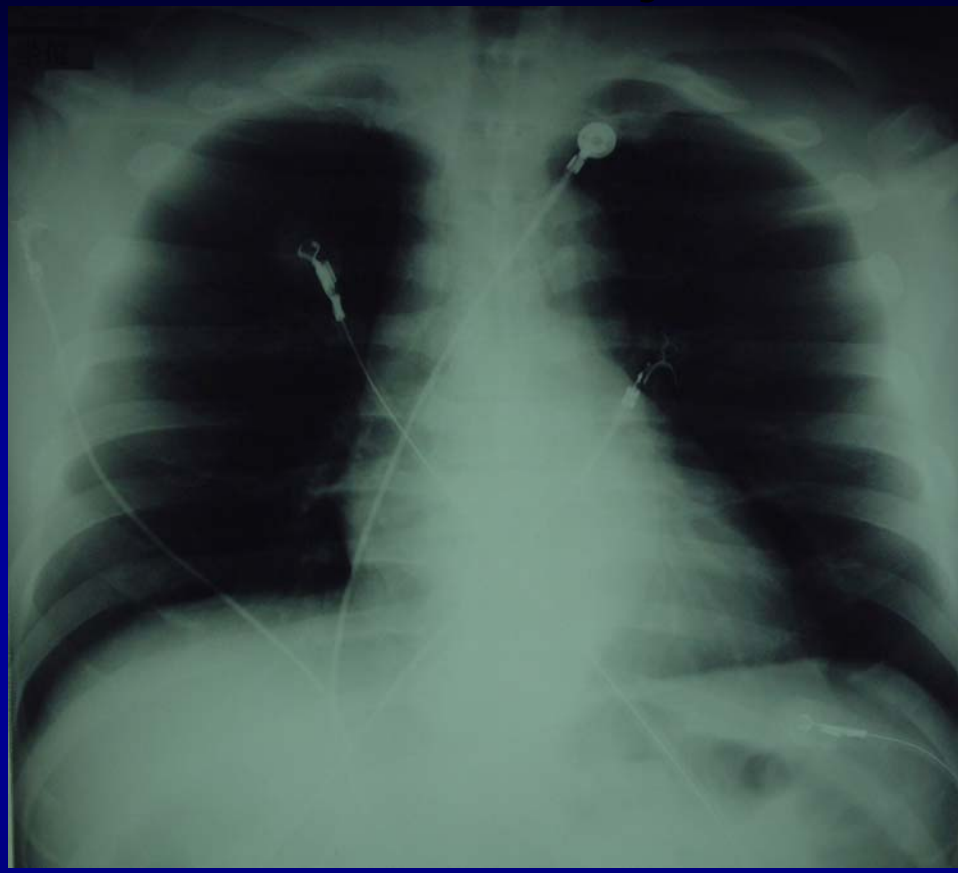
Case presentation-2;57 yrs, male

* After administration of Ca antagonist and nitrate, he was free from chest pain and arrhythmias.
* On 07/2005, he was admitted to the other hospital for the treatment of gastric ulcer.
* On 07/19/2005, he was given medication for gastric ulcer and, then, he again experienced chest discomfort.
* At noon time of next day, he suddenly passed out with his monitor ECG showing VF.
* On 07/22/2005, he was referred to our hospital for re-evaluation and treatment of VF.

Baseline 12-lead ECG



Chest X-ray

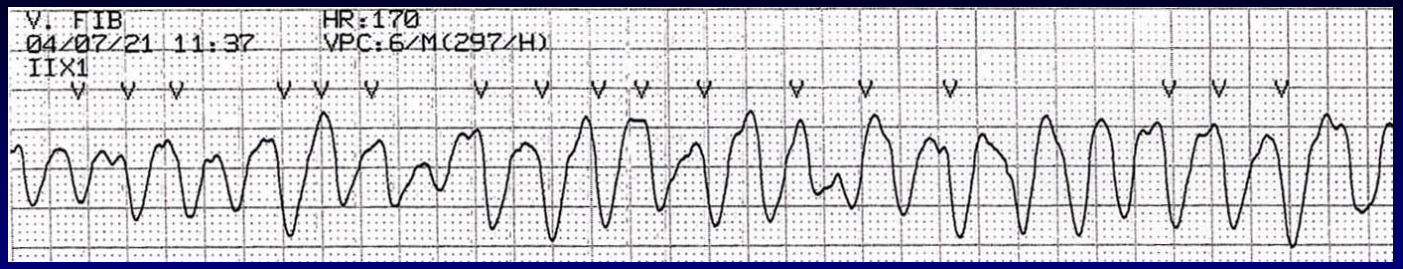


UCG

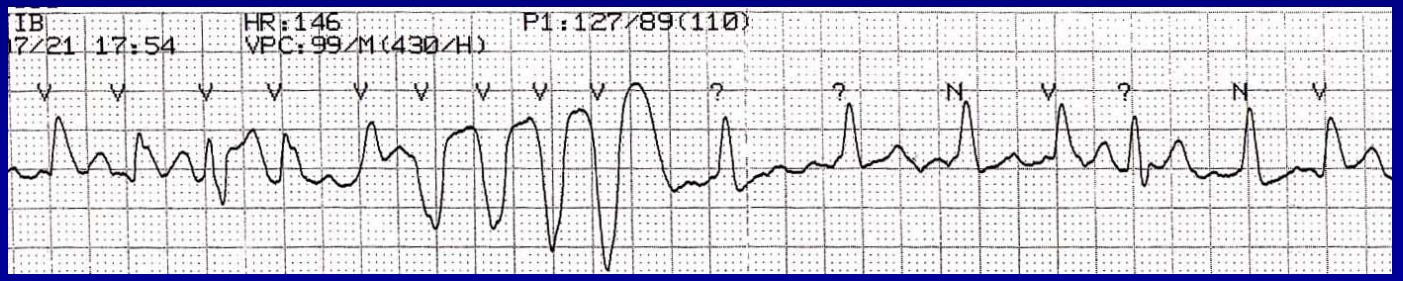
Dd/Ds; 44/30, EF=60%,
AR II°, MR I°

His baseline 12-lead ECG showed AF and faltterned T waves in V5/V6. Chest X-ray demonstrated no remarkable changes. UCG showed mild to moderate mitral (MR) and aortic regurgitation (AR).

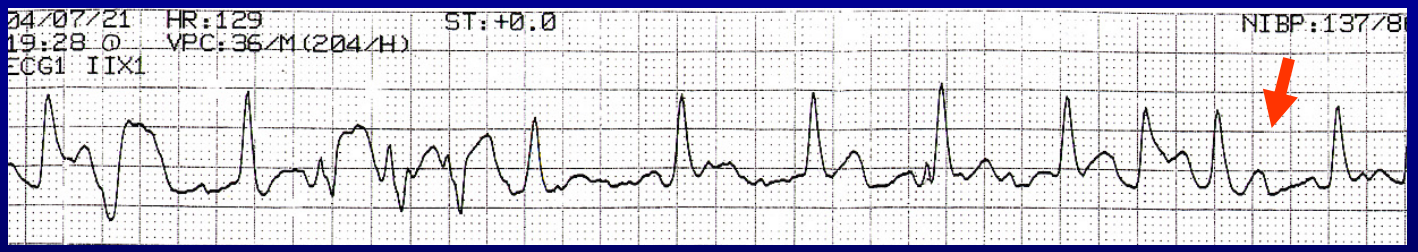
VF （11:37)



VT （17 : 54）

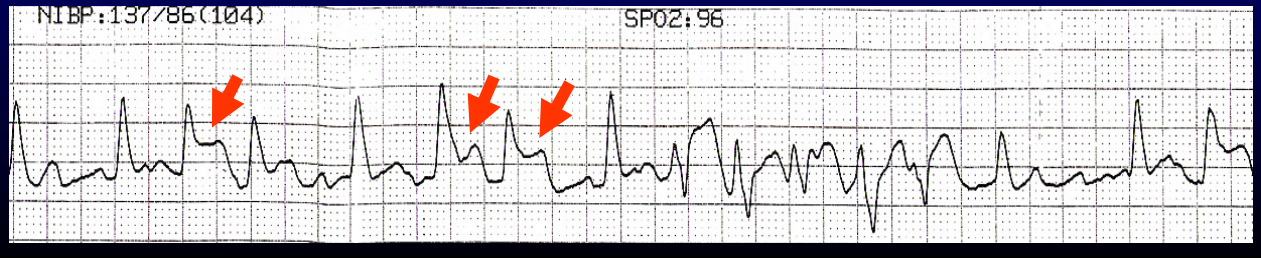


ST elevation (19:28), VT



*

* Continuous



*

His monitor ECG on the 2nd admission.

PVT and VF were seen with ST elevation.

Findings of Coronary Angiogram; No organic stenosis at baseline

[RCA]

1: spontaneous spasm of 90% occlusion at baseline

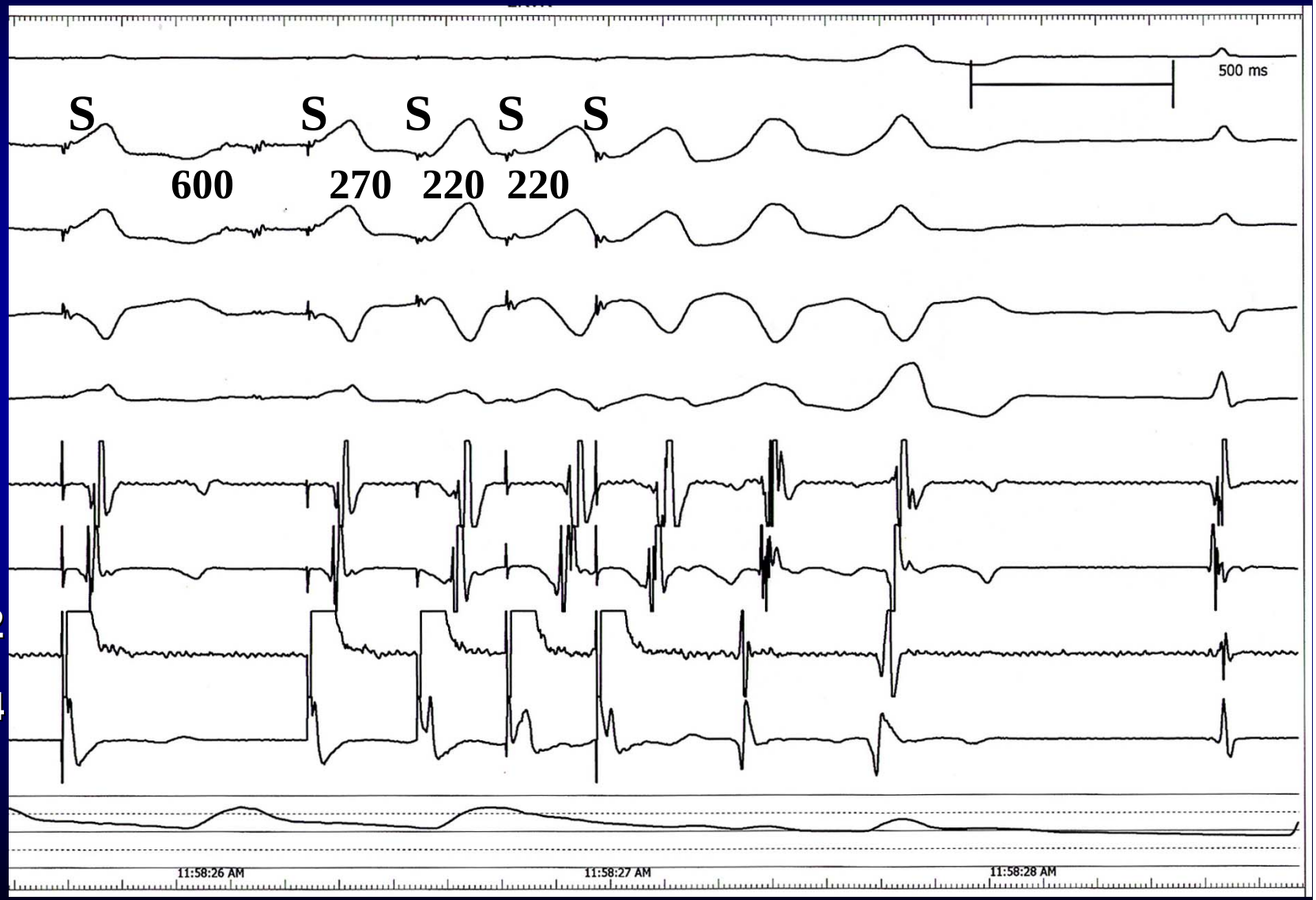
[LCA]

6: 90% and # 7: 100% occlusion

#13: 99% occlusion by 20 μg Acetylcholine (Ach)

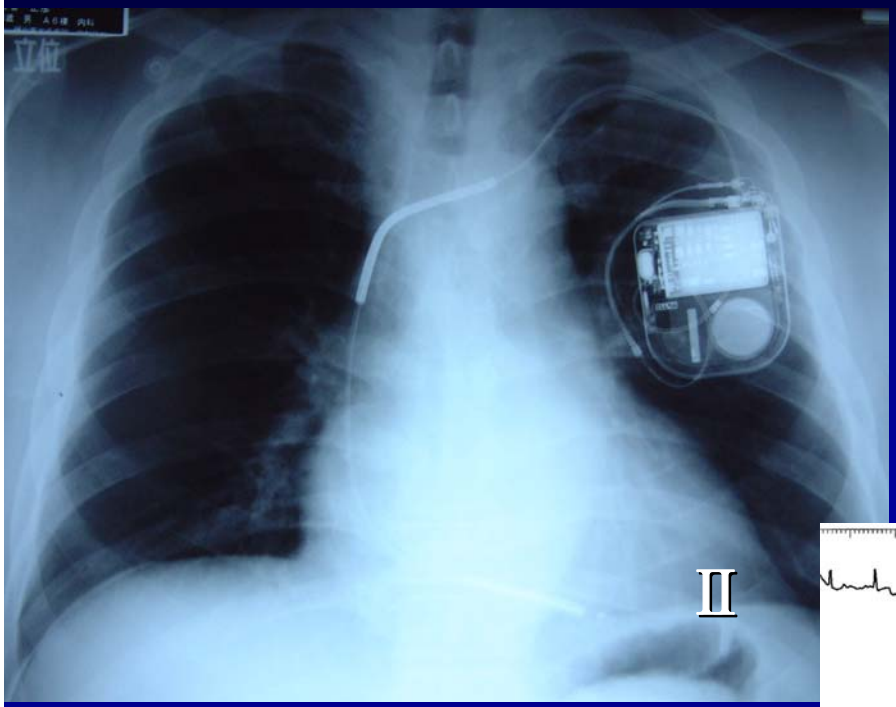
Findings of his coronary angiogram demonstrated multiple arterial spasm on both RCA and LCA during baseline and after intra-coronary administration of Ach.

Programmed ventricular stimulation at RVOT

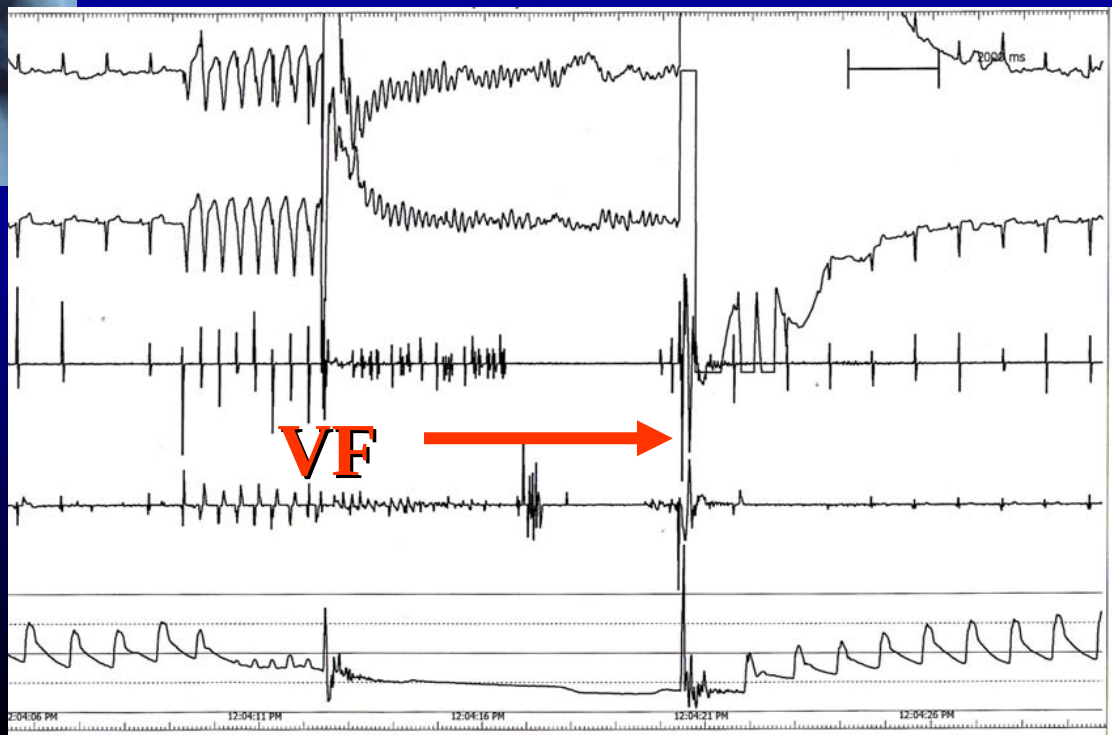


Programmed ventricular stimulation (PVS) induced two ventricular responses but failed to induce PVT.

ICD implantation



DFT: 20J B>AX



Because of frequent development of PVT/VF associated with attacks of vasospastic angina, he was implamted ICD.

Comments on PVT in vasospastic angina-1

- We studied Holter ECG recordings in 60 consecutive patients with vasospastic angina (Jpn Circ J 2001;65:519-525). Eight patients had at least one episode of PVT during Holer monitorings and remaining 52 were free of PVT.
- Ischemic ST segment elevation preceded the development of PVT in all 8 cases and 4 had silent coronary spasm. The onset of PVT was initiated with R-on-T, long-short sequence or ST wave alternans in 6 of 8 cases.

Comments on PVT in vasospastic angina-2

- During a follow-up of 73 ± 17 months, a high incidence of sudden death (2/8 cases; 25 %) in PVT group was observed, while no death was found in 52 cases of non-PVT group.
- Two cases of sudden death victims showed atrial fibrillation (AF) in baseline rhythm. Therefore, AF may carry a high risk in patients with vasospastic angina.
- (Following two slides summarize the results of this study)

Plymorphic ventricular tachycardia in patients with vasospastic angina -Clinical and electrocardiographic characteristics and long-term outcome-

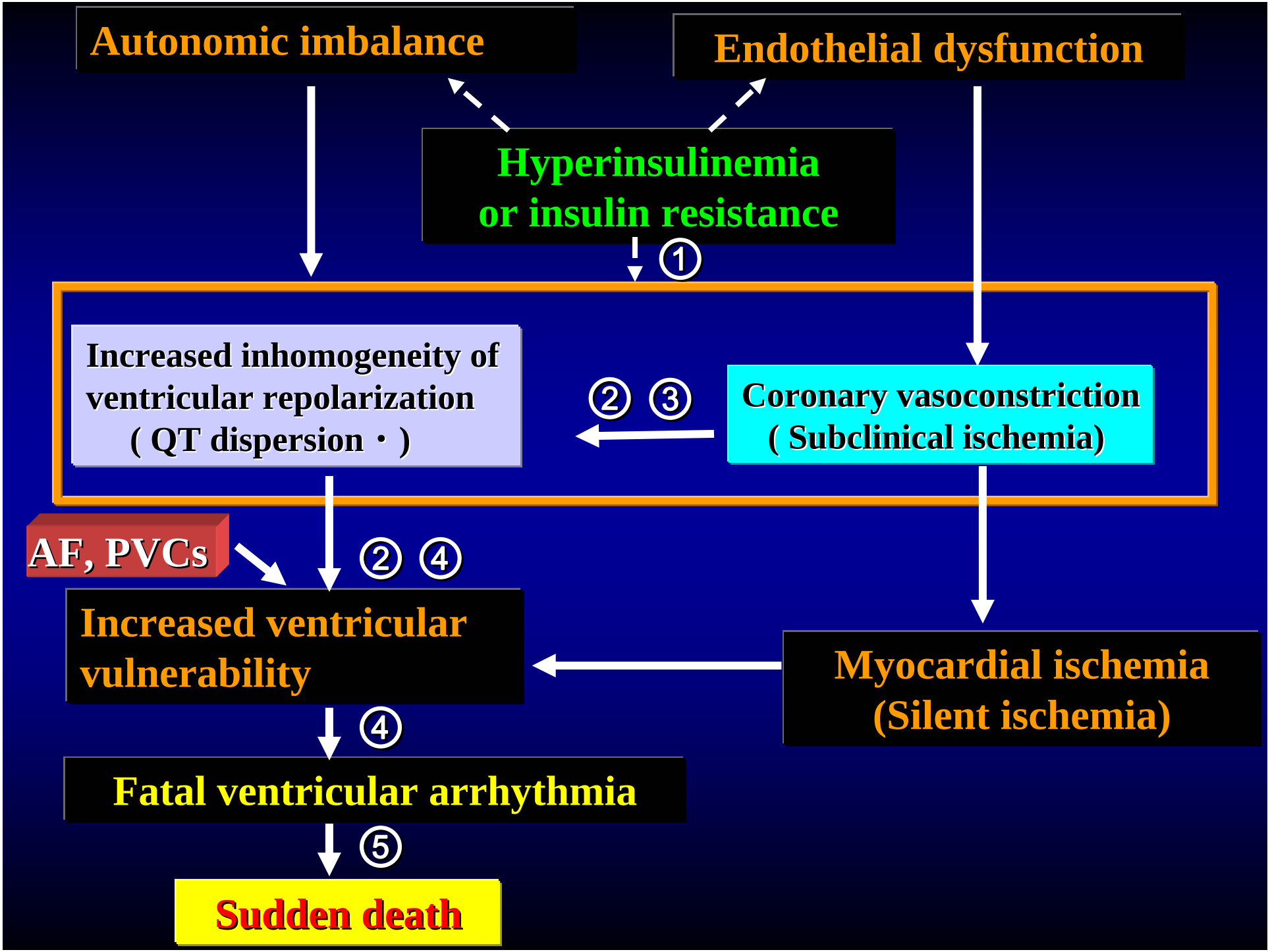
Table 1 Clinical and Electrocardiographic Characteristics of Patients With Polymorphic Ventricular Tachycardia

Patient no.	Age (years) /Sex	Date	Dominant rhythm	QT (ms)	QTc (ms)	Result of coronary arteriography	Site of coronary spasm*	Angina	ST elevation	Interval from ST elevation to onset of PVT
1	36/M	Jul. 91	Sinus	320	410	Normal	LAD(6,8), LCx(12)	+	+	60 s
2	73/M	Nov. 92	AF	340		Normal	LAD(6)	-	+	1 s
3	62/M	Apr. 92	Sinus	360	440	Normal	LAD(6)	-	+	105 s
4	60/M	Oct. 93	Sinus	380	414	Normal	LAD(6)	+	+	135 s
5	67/M	Apr. 93	Sinus	380	380	Normal	LAD(6)	+	+	180 s
6	52/M	Apr. 93	Sinus	420	400	Normal	LAD(8), LCx(13), RCA(2)	+	+	165 s
7	60/M	Nov. 94	AF	400		Normal	LAD(6)	-	+	75 s
8	44/M	Sep. 95	Sinus	400	417	Normal	LAD(7)	-	+	105 s

Jpn Cir J, 2001; 65: 519-525

PVT in vasospastic angina -2

R on T	Long–short sequence	T wave alternans	Tdp	Follow-up period (months)	Medication	Outcome	Cause of death
+	+	+	+	95	Diltiazem 90 mg, nicorandil 15 mg	Survived	
–	–	–	–		Nifedipine 40 mg, ISDN 60 mg	Died at 5 months	VF
+	+	–	+	86	Nifedipine 40 mg, ISDN 60 mg	Survived	
–	+	+	–	68	Diltiazem 90 mg, ISDN 60 mg	Survived	
+	–	–	–	74	Diltiazem 120 mg, ISDN 60 mg, nicorandil 15 mg	Survived	
–	–	–	–	74	Diltiazem 90 mg, nifedipine 40 mg, ISDN 80 mg	Survived	
+	+	–	+		Diltiazem 90 mg, ISDN 60 mg	Died at 18 months	VF
+	+	–	+	45	Diltiazem 120 mg	Survived	


Autonomic imbalance
Endothelial dysfunction
Hyperinsulinemia
or insulin resistance
①
Increased inhomogeneity of
ventricular repolarization
(QT dispersion・)
② ③
Coronary vasoconstriction
(Subclinical ischemia)
AF, PVCs
② ④
Increased ventricular
vulnerability
Myocardial ischemia
(Silent ischemia)
④
Fatal ventricular arrhythmia
⑤
Sudden death

Our proposed schema contributing to PVT/VF (Fatal ventricular arrhythmia) and SCD.

Numbers indicate references cited in next slide.

References

- ① J Am Coll Cardiol, 1996 ； 27:1458-63
- ② Circulation, 1998; 98: 435-440
- ③ Am J Cardiol, 1999; 84:807-810
- ④ Am J Cardiol, 1996;77:355-360
- ⑤ Jpn Cir J, 2001; 65: 519-525