



## Palpitations, diagnostic work-up, pacemaker therapy

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“How do device-detected  
(asymptomatic) arrhythmias  
translate into therapeutic  
strategy?”

# Agenda

- When to monitor invasively ?
- How common is asymptomatic AF in patients with an implantable device ?
  - Without prior TE event
  - With a prior TE event
- What are the consequences in regard to anticoagulation in each case ? (prediction vs prevention)

## Patients with suspected but undiagnosed atrial fibrillation

ent monitoring strategies.<sup>3,34–37</sup> More intense and prolonged monitoring is justified in highly symptomatic patients [European Heart Rhythm Association IV (EHRA IV)—see Section 3.6], patients with recurrent syncope, and patients with a potential indication for anticoagulation (especially after cryptogenic stroke).<sup>34,38</sup> In selected patients, implantation of a leadless AF monitoring device may be considered to establish the diagnosis.<sup>39</sup>

*Source: 2010 ESC AF management guideline*

## Asymptomatic AF in the CIED pt

- Subgroup from MOST trial (DDD vs VVI pacing)
- n=312 patients with AHRE, from total 2010 in study (15.5%)
- AHRE >220 bpm > 5min
- With and without history of AF
- FU 6 yrs

	HR for AHRE +	p
All mortality	2.48	0.0092
Death/nonfatal stroke	2.79	0.0011
AF	5.93	0.0001

*Glotzer. Circ 2003;107:1614-9*

## Asymptomatic AF in the CIED pt

- TRENDS trial
- n=2486 pts,  $\geq 1$  risk factor for stroke
- With and without history of AF
- ICD or PM implant
- AT/AF burden as longest duration episode last 30 days
- FU 1.4 yrs

AT/AF burden	TE risk/yr (%)	
Zero	1.1	
Low (< 5.5 hrs)	1.1	
High ( $\geq 5.5$ hrs)	2.4	p=0.06

*Glutzer. Circ Arrhythmia Electrophysiol 2009;2:474-80*

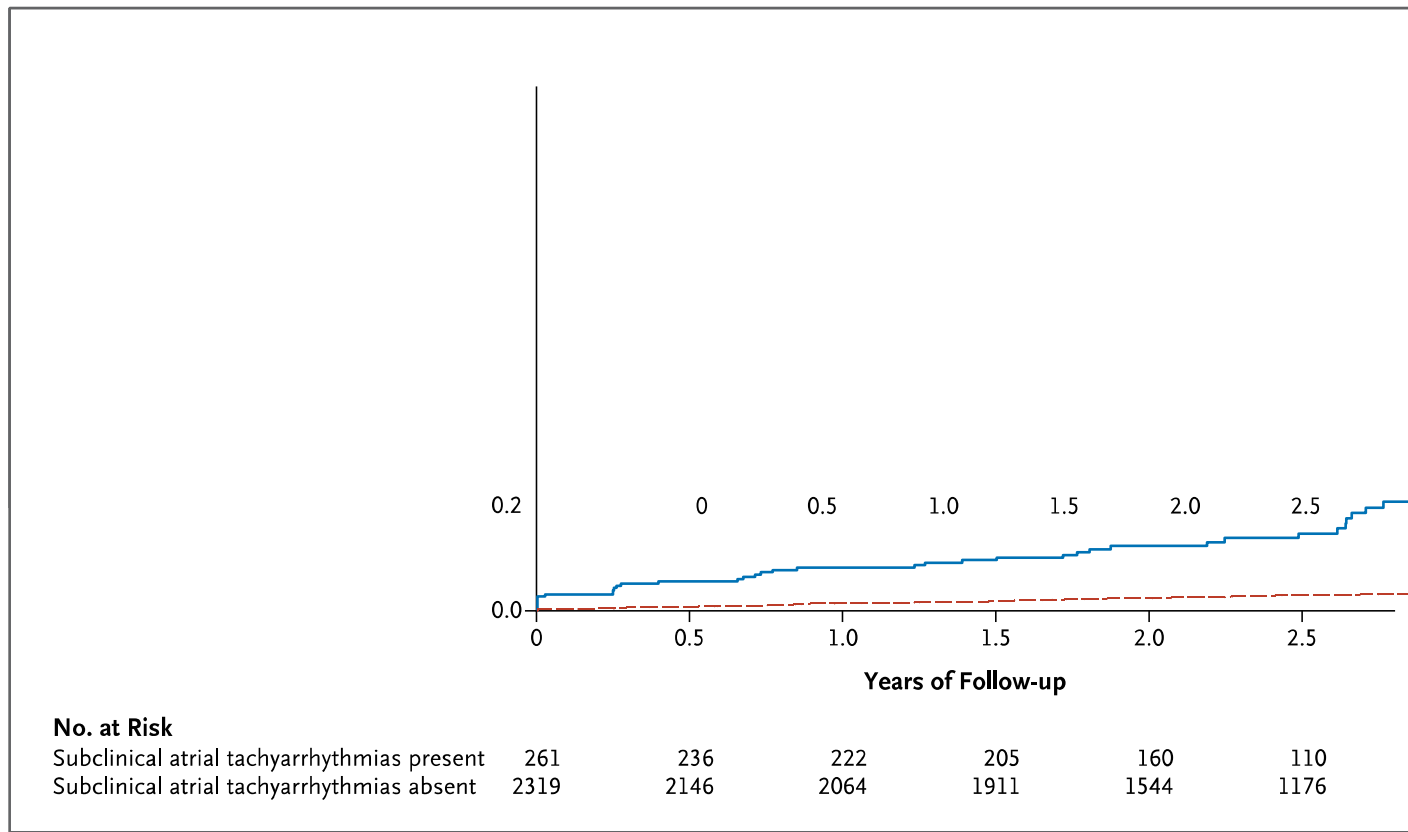
## Asymptomatic AF in the CIED pt, no prior history of stroke

- ASSERT trial
- n=2580, >65 yrs
- No history of AF
- Recent PM / ICD
- 3 mo monitoring for subclinical atrial arrhythmias
  - Atrial rate >190 bpm
  - For > 6 min
- 2.5 yrs FU, primary endpoint ischemic stroke / SE

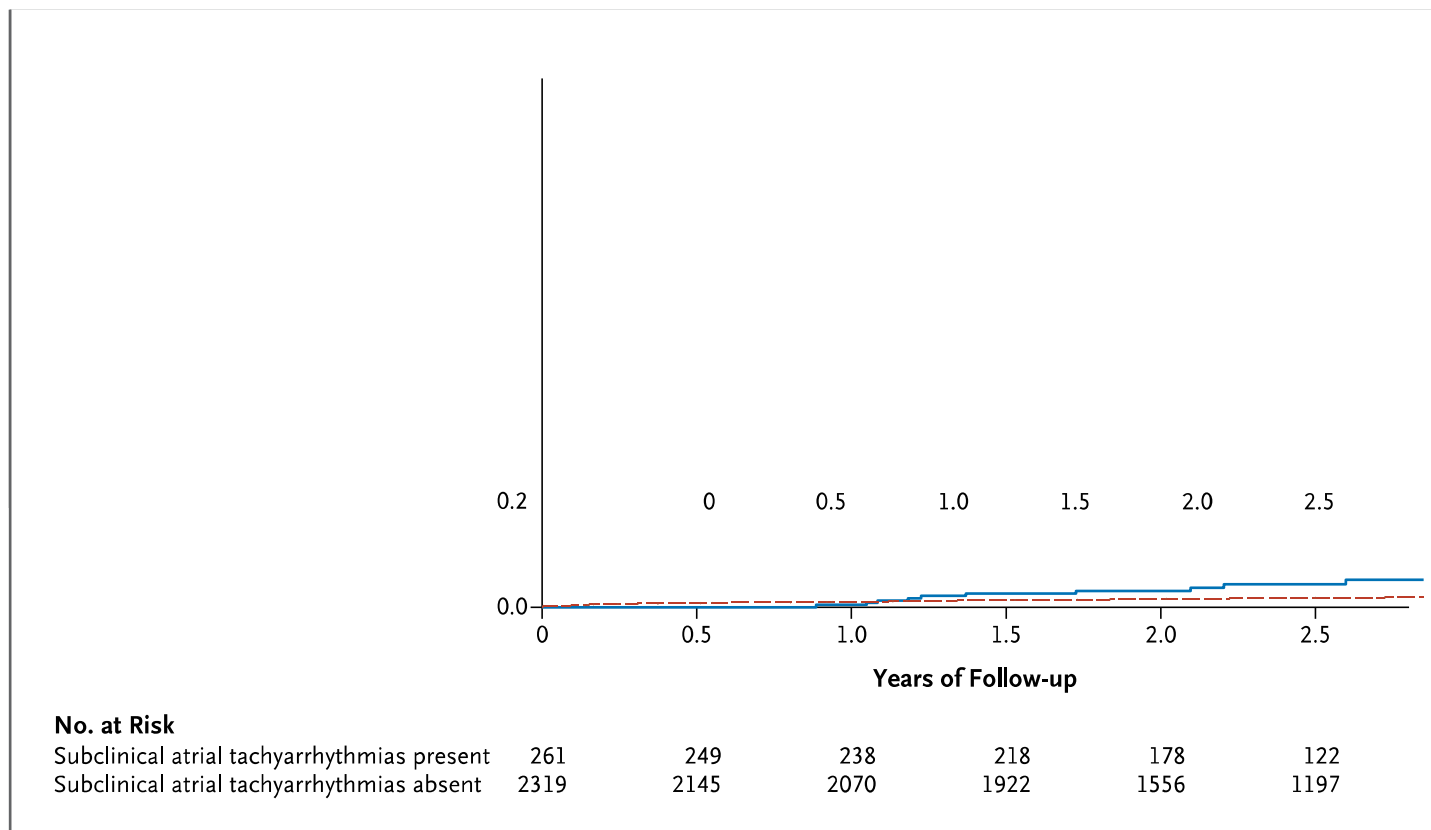
*Healy. NEJM 2012;366:120-9*



# ASSERT, results



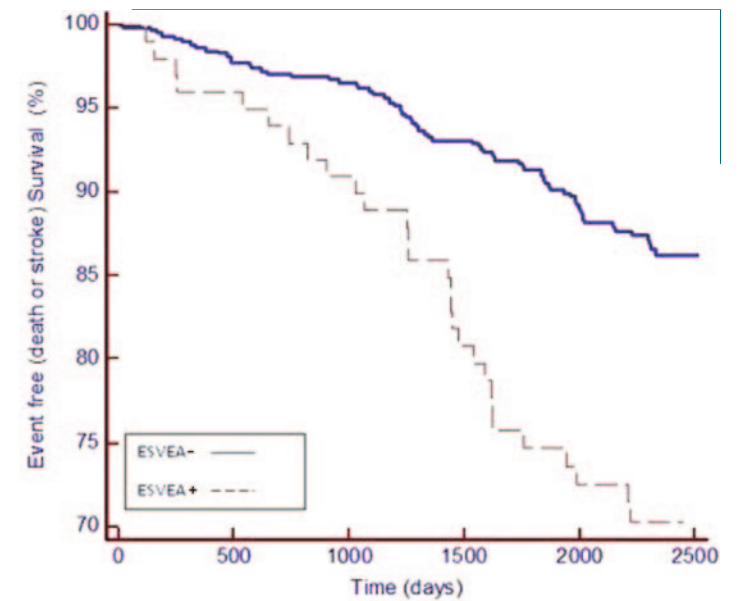
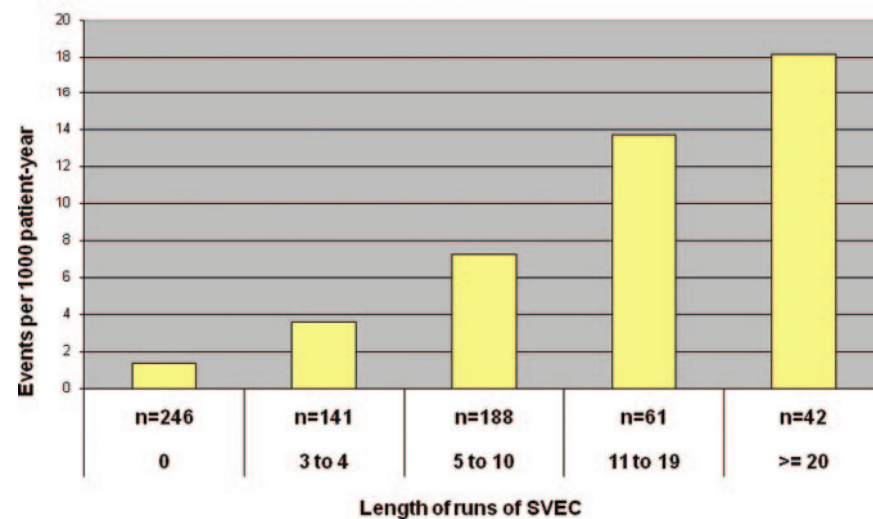
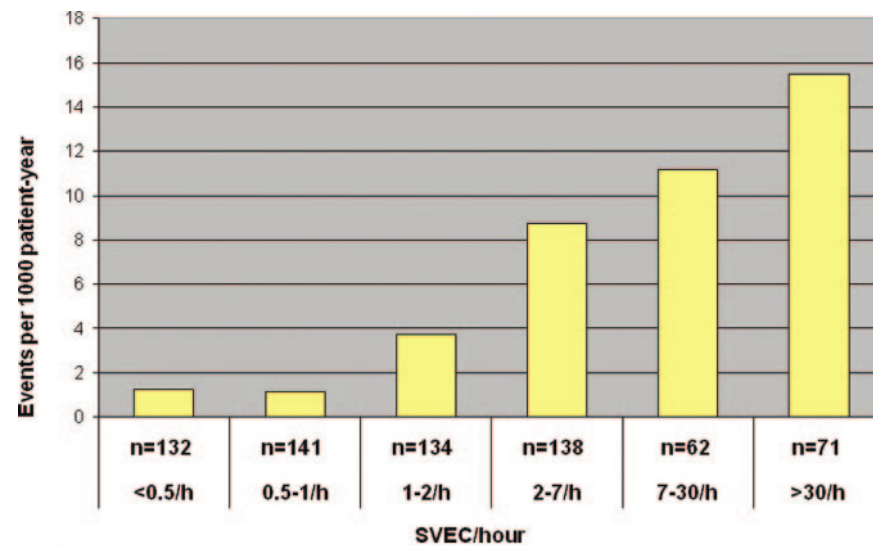
# ASSERT, results



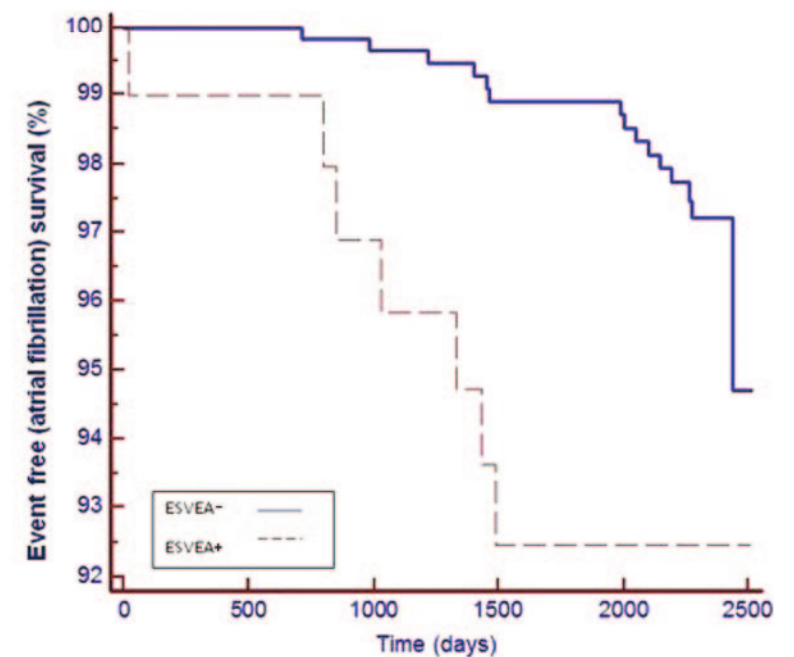
# Copenhagen Holter Study

- 678 healthy subjects, 55-75 yrs
- No history of AF, stroke, CV disease
- 48hr Holter
- Excessive supraventricular ectopy (ESVA)
  - $\geq 30$  PAC/hr
  - Any run  $\geq 20$  beats
- FU mean 6.3 yrs
- Primary endpoint death or stroke

*Binici. Circulation 2010;121:1904-11*



Number at risk: ESVEA -	579	566	559	539	500	3
ESVEA+	99	95	90	80	66	0



Number at risk: ESVEA -	579	568	560	538	500	4
ESVEA+	99	96	92	79	70	0

## Copenhagen Holter Study, results

- 14.6% had ESVA
- HR for CVA / death: 1.64 (p=0.036)
- HR for stroke: 2.79 (p=0.014)
- HR for admission with AF: 2.78 (p=0.033)

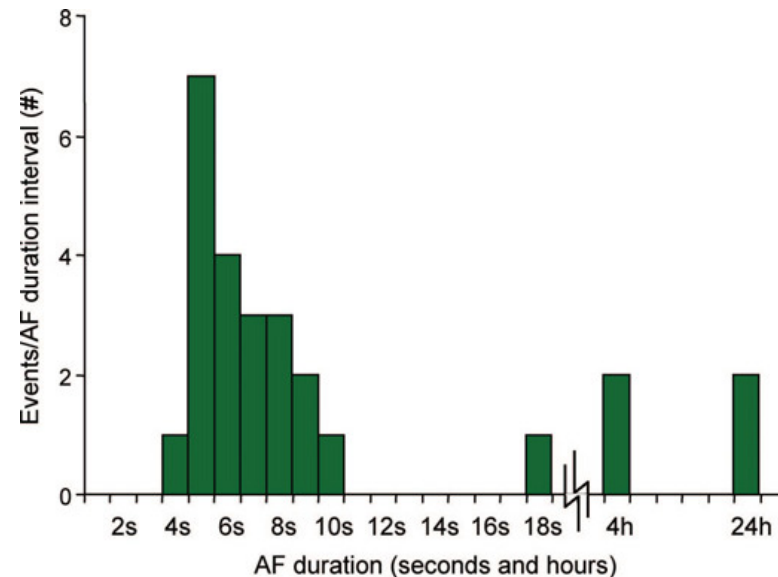
## Asymptomatic AF in the CIED pt, *with a prior history of stroke*

- TRENDS prior stroke subgroup analysis
- 319 patients with TE event
- Excluded: 80 with history of AF, 56 on warfarin, 20 on AAD
- Leaving 163 patients for analysis
- Newly detected AT/AF in 28% over 1.1 yrs FU

*Ziegler. Stroke 2010;41*

## Asymptomatic AF in the pt *with* a prior history of stroke

- 56 pts, cryptogenic stroke
- Mobile outpatient telemetry 5-21 days
- 23% newly diagnosed AF at a median 7 days
- 85% < 30sec duration



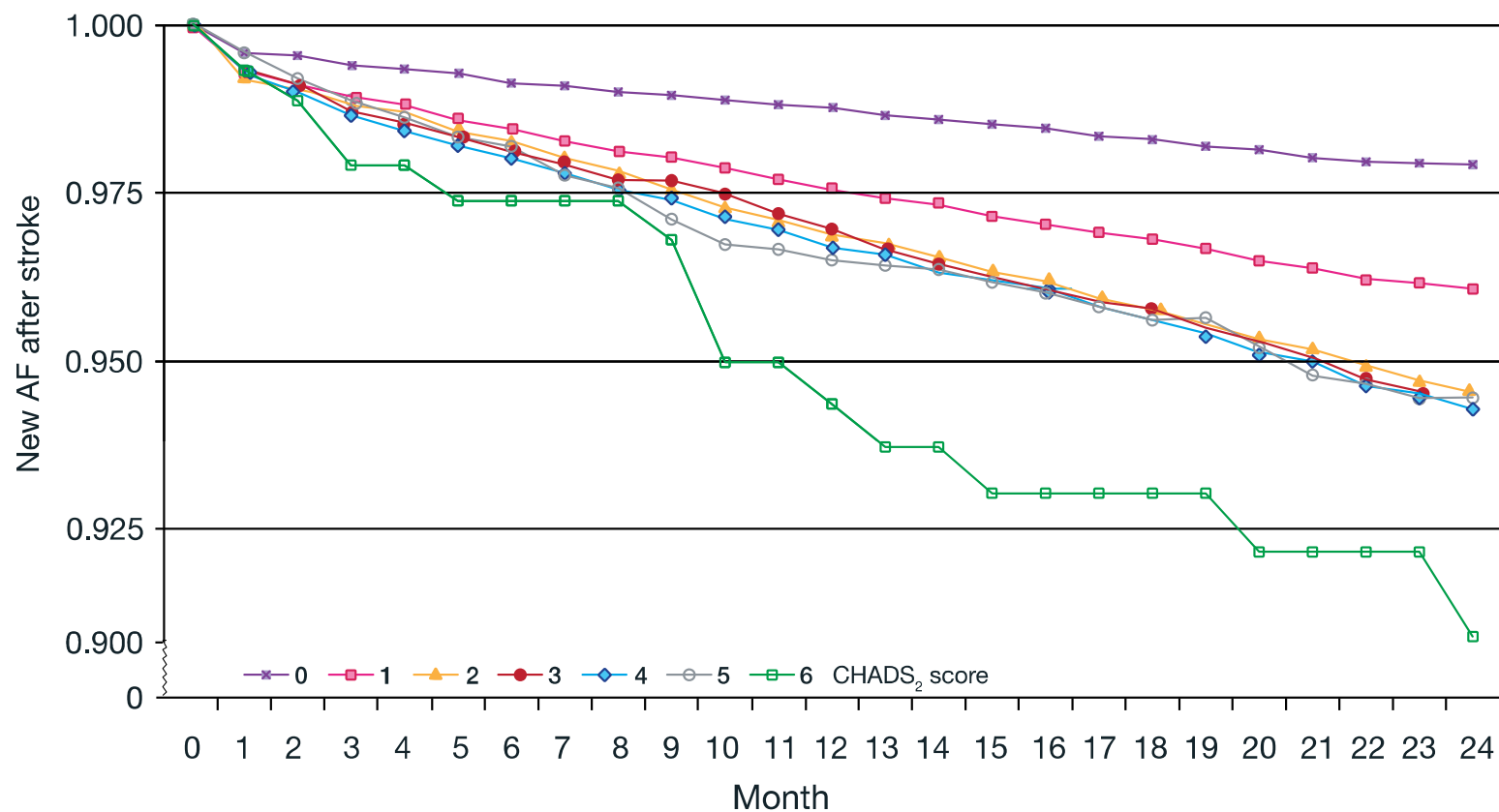
*Tayal. Neurology 2008;71:1696-1701*

## Diagnosis of AF *after* stroke

- Swedish stroke registry
- n=57636 pts with nonfatal stroke
- No prior AF diagnosis
- 2.2 yrs FU
- Incidence of hospitalisation for AF 2769pts (4.8%)
- Stratified per CHADS<sub>2</sub> score



# Swedish Stroke Registry



Henriksson. Clin cardiol 2011;34:309-16

## Work in progress

- CRYSTAL AF trial
- Randomized prospective multicentre study in 450 pts with cryptogenic stroke
- 1:1 standard monitoring:Reveal XT
- Primary endpoint time to detection of AF within 6 mo after stroke
- FU at least 12 months
- Study completion expected late 2012

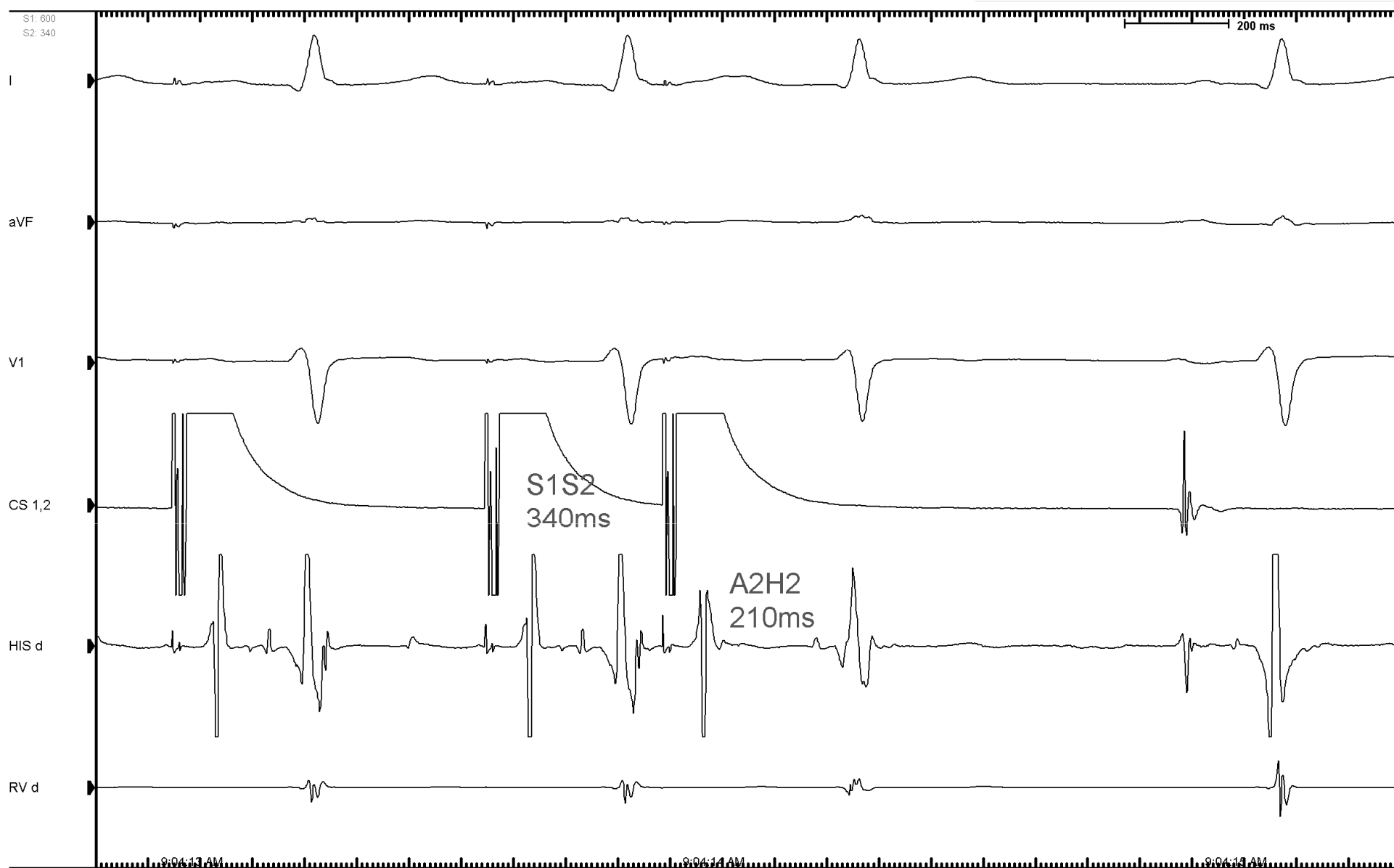
*Sinha. Am Heart J 2010;160:36-41*

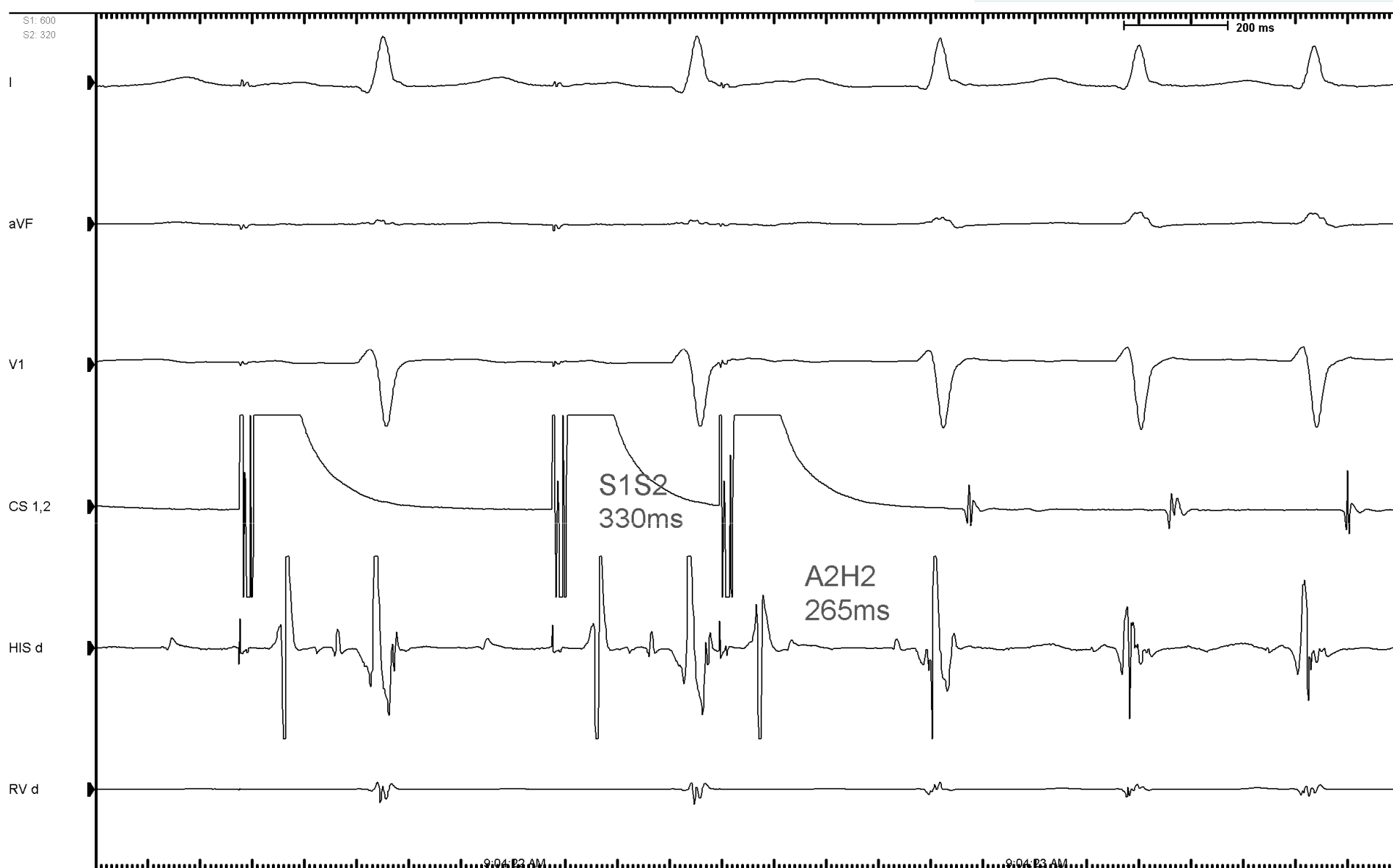
## Summary

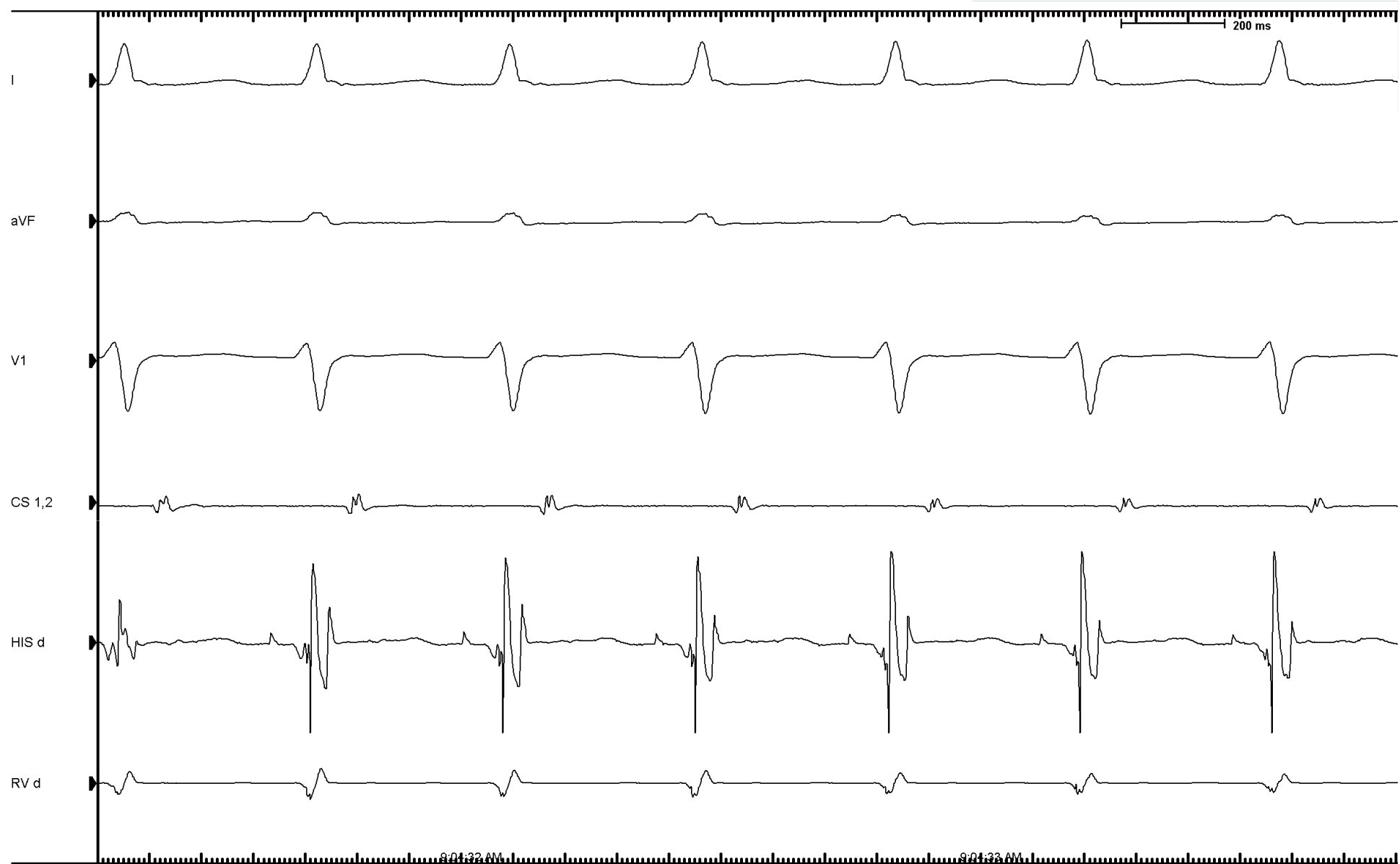
- Even very brief, sub-clinical episodes of AF are associated with increased stroke risk
- They are markers for occurrence of clinical AF
- Extended monitoring post cryptogenic stroke is warranted, especially in high risk populations
- Screening in (elderly) high risk population *without* prior TE event?
- More aggressive anticoagulation strategies may be warranted in these pt populations

## Case

- 39 year-old male
- Structurally normal heart and resting EKG
- Highly symptomatic, documented PAF since early adulthood
- Referred for PVI due to symptoms + drug side-effects



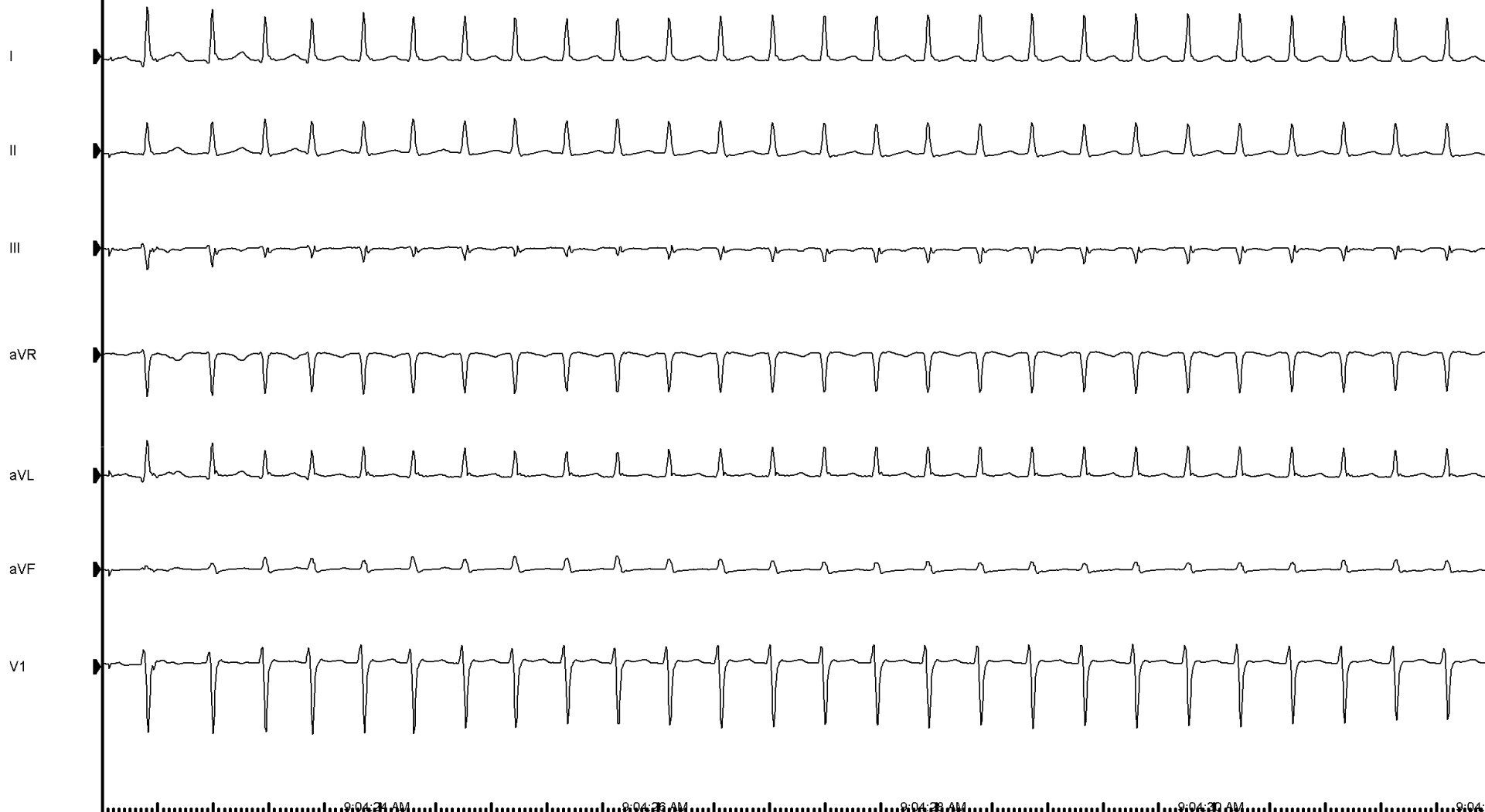




S1: 600  
S2: 320

1000 ms

AVNRT CL 360ms





## Other SVT's / non-PV foci as triggers for AF

- n=409 pts referred for PVI
- 55±9 yrs
- All underwent EP study and ablation aimed at any other arrhythmia
- 7.6% inducible SVT
  - Flutter 3.7%
  - AVNRT 1.7%
  - AVRT 1.2%
  - AT 1.0%
- AF recurrence rate significantly higher in flutter than all other (53%, p=0.03)

*Katritsis. Europace 2007;9:785-9*

## Other SVT's / non-PV foci as triggers for AF

- 257 pts referred for PVI
- 10.1% inducible SVT, ablated
  - 4.7% AVNRT
  - 3.5% AVRT
  - 1.9% AT
- Younger than rest of cohort (43 vs. 57 yrs)
- 7.7% AF recurrence rate

*Sciarra. Europace 2010;12:1707-12*

## AF / WPW

- 116 pts with PAF, referred for ablation of an AP
- AF recurrence 12% < 50 yrs, 35% > 50, 55% > 60
- Vs 4% in control group of 100 pts without prior AF

*Dagres. Eur Heart J 2001;22:423-7*

## AF / AVNRT

- 629 referrals for PVI
- AVNRT inducible in 4.3%
- Younger at onset of symptoms than rest of cohort (37 vs. 48 yrs)
- Ablation: 14/27 pts PVI and SP, 13/27 SP only
- FU 21.4 mo
- 12/13 pts AF-free off drugs after only SP ablation

*Sauer. Circulation 2006;114:191-5*

# Conclusions

- Asymptomatic AF is highly prevalent in CIED pts
- It predicts TE events
- This should have a profound effect on management of (preventive) anticoagulation therapy
- In addition to utilisation of ILRs in high risk patients
- In patients referred for PVI, the arrhythmia you see is not always what you get
- This should also be reflected in diagnostic and therapeutic approach to the PVI candidate



