# Mapping and Ablation of VF

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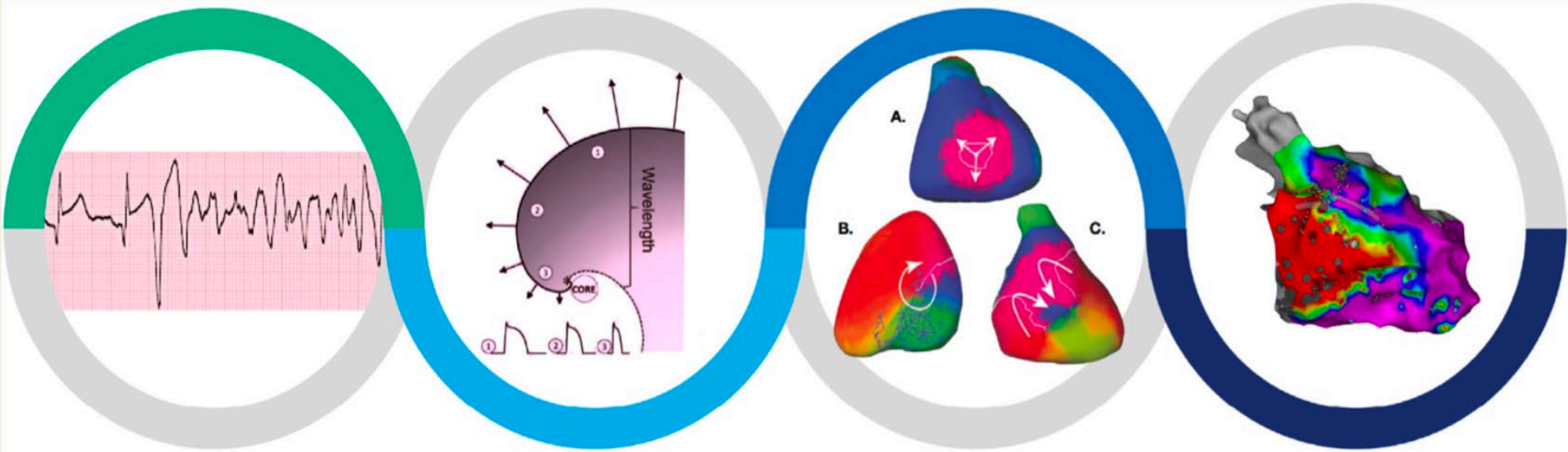
# Ventricular Fibrillation

- VF major cause of SCD[1]
- Underlying etiology may be
  - -SHD (scar-mediated)
  - -Channelopathies
  - -Idiopathic (IVF): 35% of SCD adults <35y age
- ICD mainstay of $R^x$ (primary, secondary)[2]
- ICDs do not prevent VF
  - Up to 20% may experience recurrent VF
- Catheter ablation for VF can prevent VF recurrence
  - Emerging role
- Expanding body of knowledge on VF rotors may allow novel targets for ablation

[1]John Lancet 2012; [2]AVID NEJM 1997; [3]Huang Prog CV diseases 2008

# VF Mechanism




**INITIATION**

- Triggering PVCs
- RVOT
- Purkinje
- Papillary Muscle
- VT

**TRANSITION**

- APD heterogeneity
- Steep APD restitution
- Alternans
- CV Restitution
- Wavebreaks

**MAINTENANCE**

A. Focal Sources
B. Motor Rotor(s)
C. Figure of 8 Re-entry

**EVOLUTION**

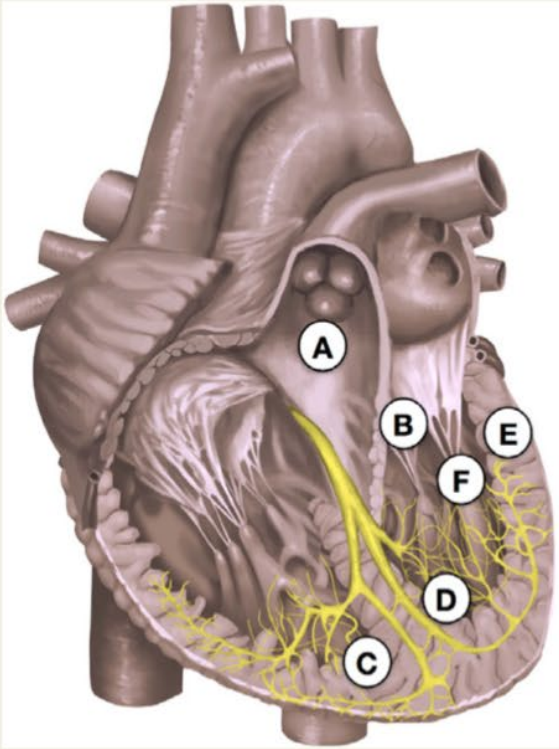
- Electrical Remodelling
- Anchoring of Rotor(s) to scar

Cheniti et al Curr Treat Options Cardiovasc Med 2017

4 distinct phases of VF
Recent work highlighted each phase may be a target for ablation

# Initiation

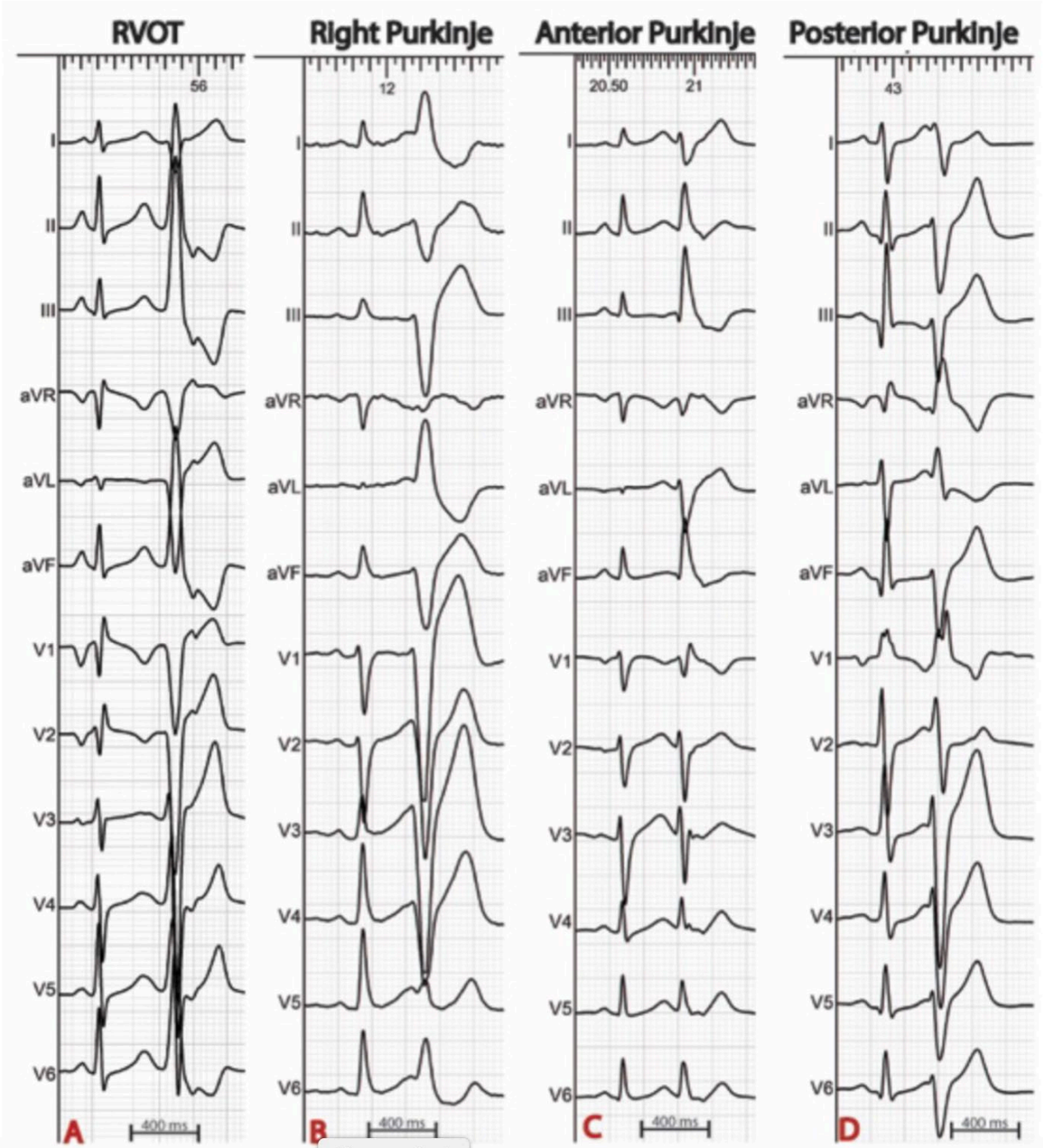
- Triggering PVC or re-entrant VT
- PVCs fall within ‘vulnerable period” (R on T)
- Short coupling interval <300ms (“short coupled variant Torsades”)
- Underlying PVC mechanism
  - -abnormal automaticity (def $Ca^{2+}$ regulation in SR)
  - -triggered activity (EAD or DAD due to $Ca^{2+}$ overload, usu Purkinje mediated–ischemia, electrolyte imbalance exacerbate)
  - -Purkinje re-entry
  - -Phase 2 re-entry
- Purkinje origin in 93% of cases of IVF
- Within areas of scar/border zones in patients with SHD




**Location of PVC triggers**

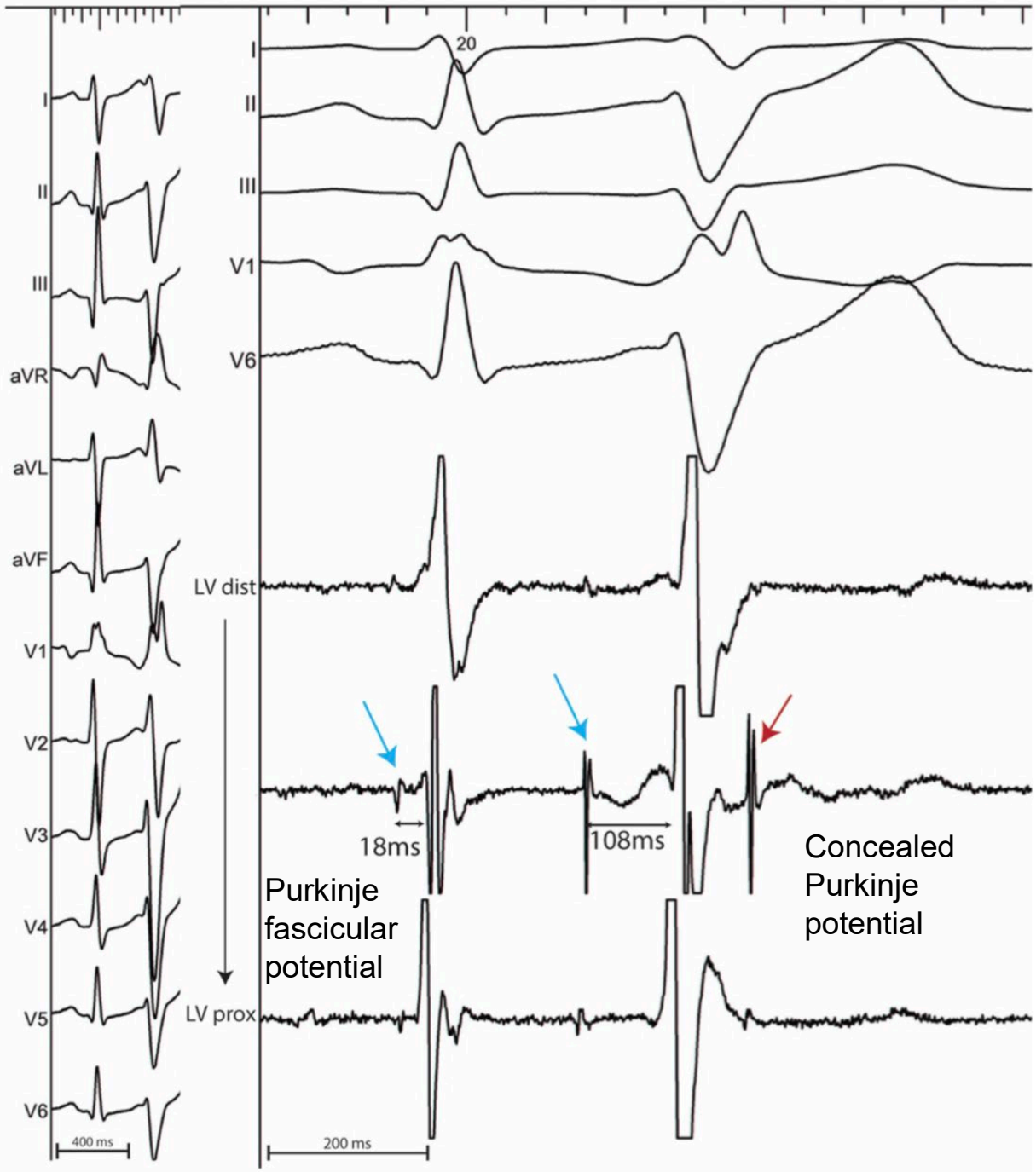
| Anatomical Site | | n (%) | Conditions |
|---|---|---|---|
| A | **RVOT** | 13 (10%) | IVF, BrS |
| B | **LVOT** | 9 (7%) | IVF, DCM |
| C | **Purkinje** | 73 (59%) | IVF, LQTS, ER, IHD, BrS, DCM |
| C | *RV-Purkinje* | 15 | |
| D | *LV-Purkinje* | 53 | |
| D | *Both-Purkinje* | 5 | |
| E | **Myocardium** | 16 (14%) | LQTS, ER, IVF, DCM |
| F | **Papillary Muscle** | 13 (10%) | IVF, DCM |

Anderson, Kumar, Lee HLC 2019




Cheniti et al
Curr Treat
Options
Cardiovasc
Med 2017

Screenshot

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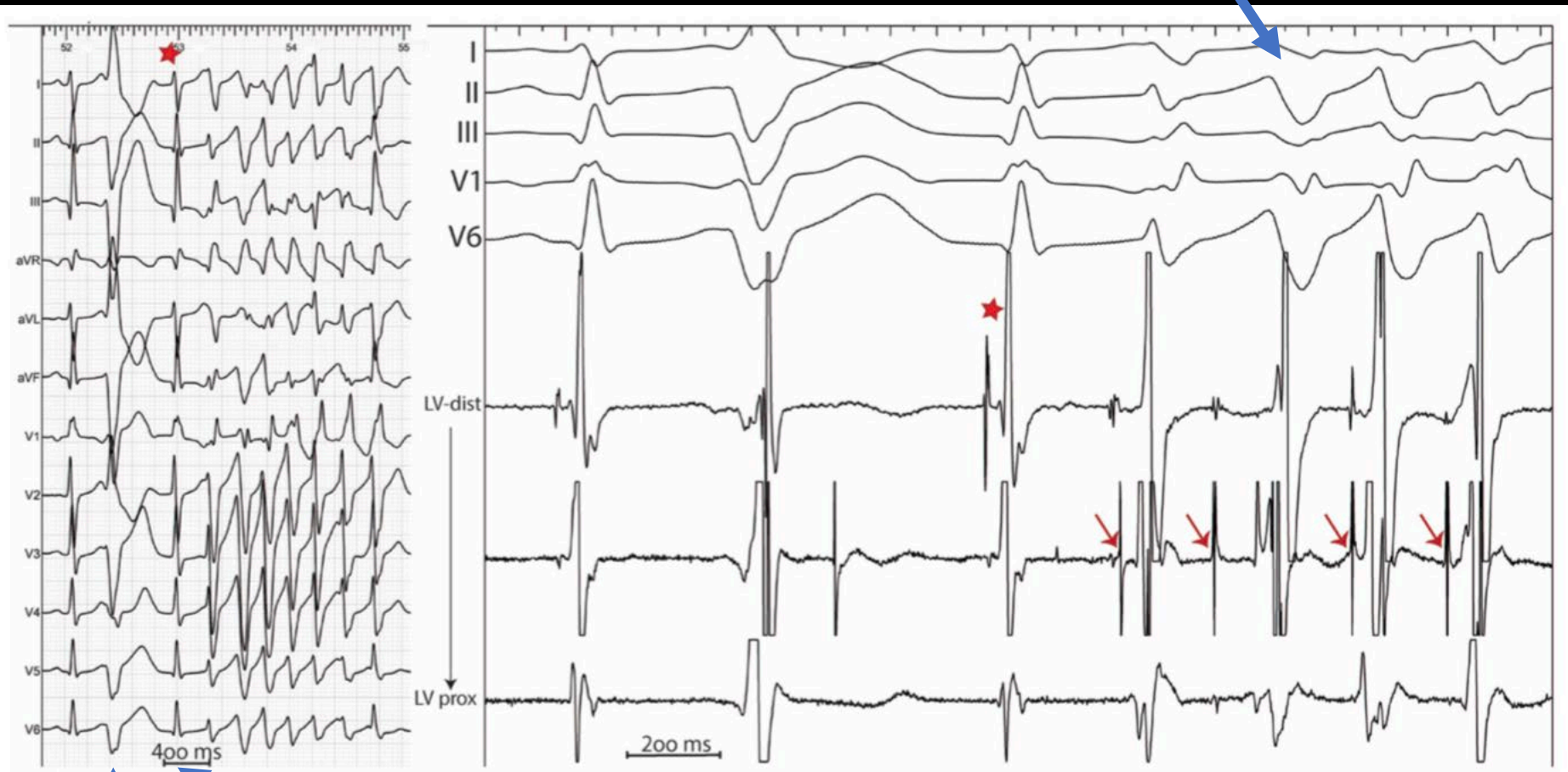
Modification in PVC morphology due to complex arborization of Purkinje network

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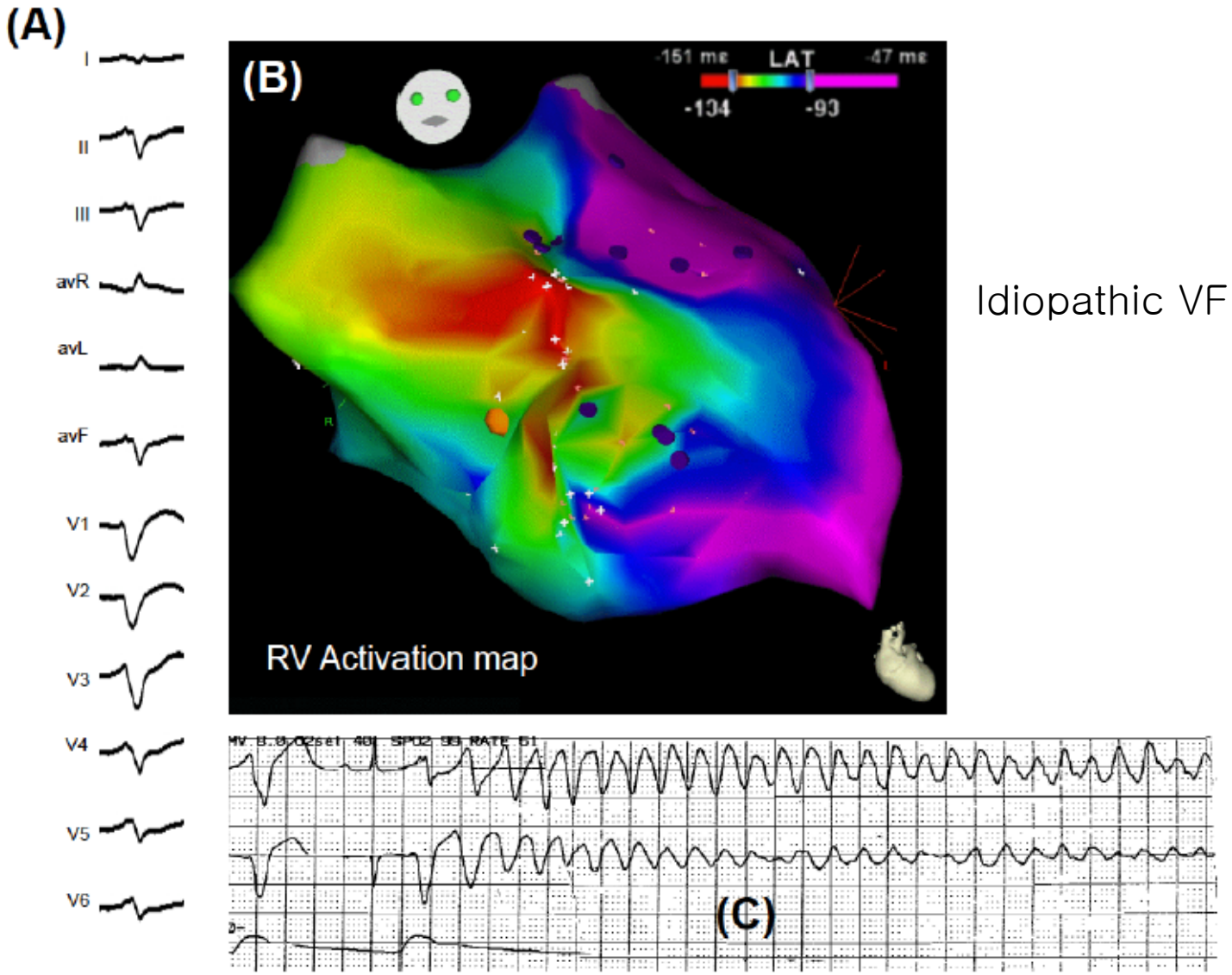
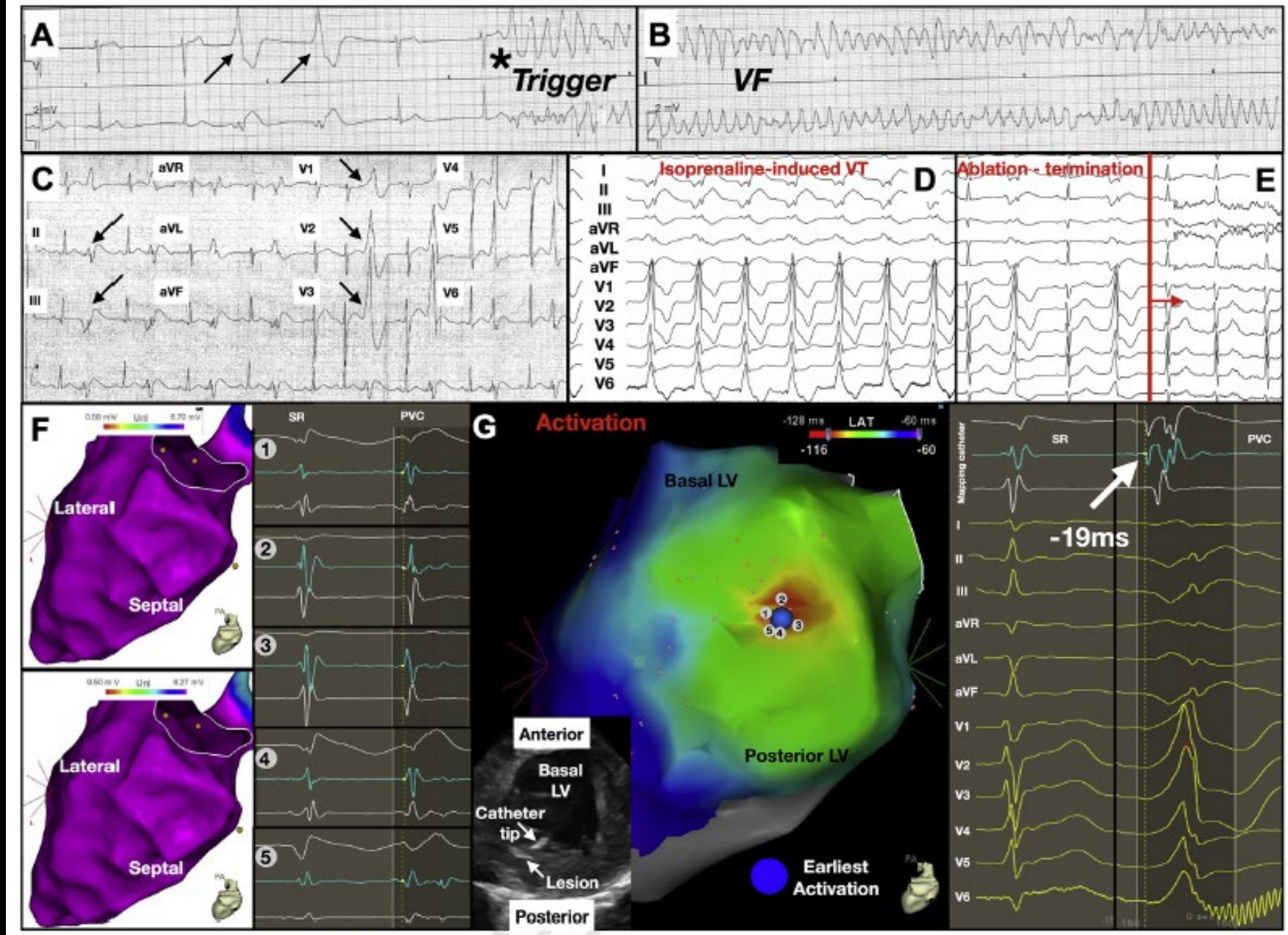
Triggering PVC

Penetration into Purkinje network

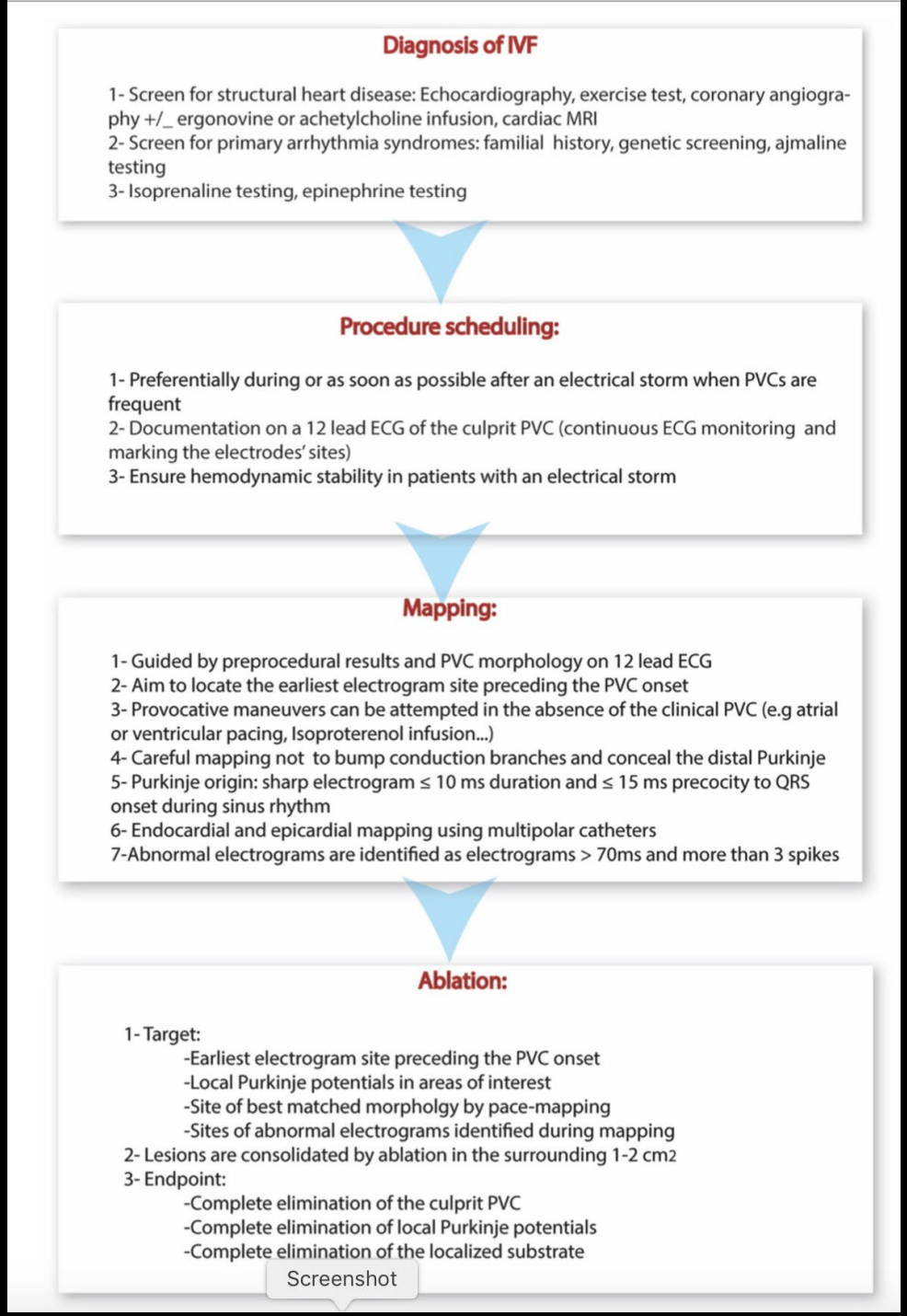



Idiopathic VF

Kumar et al Circulation EP 2015




Anderson, Kumar JACCEP 2017




Decapolar catheter (HPS)

Multi-spine (maximise mapping)

Paucity of PVCs: Pace mapping (unreliable Purkinje), ECGi

Variable morphologies may occur (variable exits)

Recurrent VF may occur (ECMO, IMPELLA may be needed)

Cheniti et al Curr Treat Options Cardiovasc Med 2017

# Outcomes of PVC-triggered VF

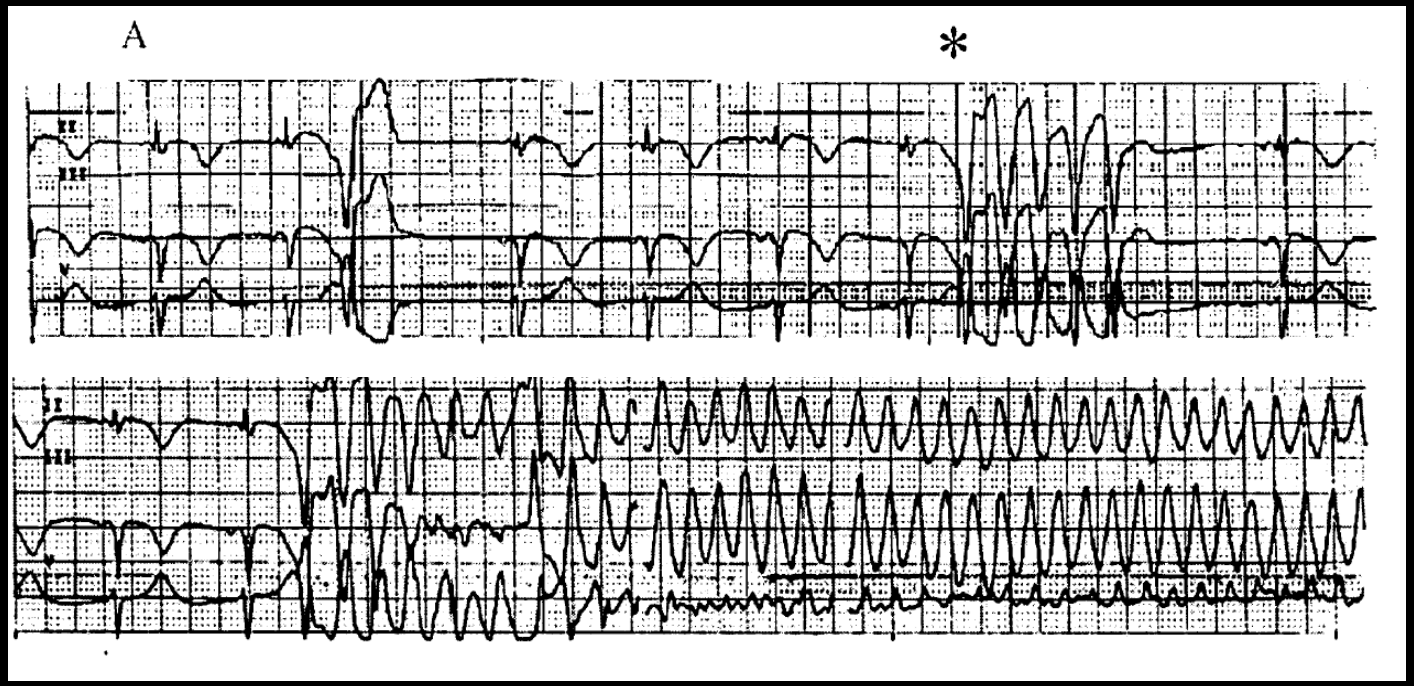
- Acute success almost universal in most published series
- Meta-analysis of published studies 5y follow up[1]
  - -VF recurrence rate 31%
  - -Mortality 3.1%
- No association bw baseline inducibility and recurrence

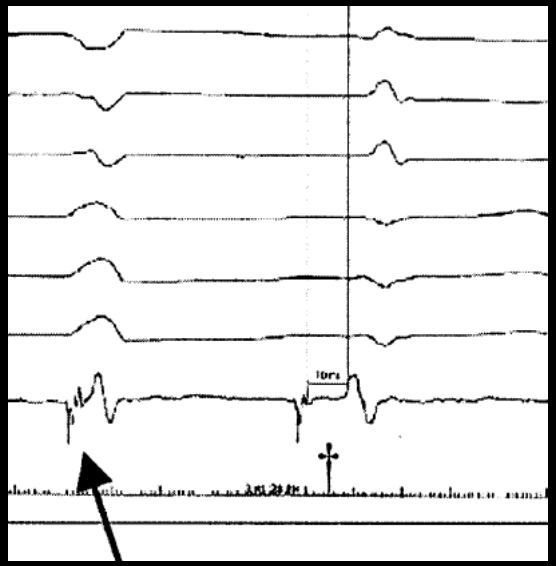
Ozaydin JCE 2015

# Importance of Purkinje system in initiation of VF

- Purkinje arrhythmogenicity (electrolyes, drugs, ischemia, HF)
- Ischemia= change in K+, $Ca^{2+}$ enhanced DADs, EADs, impaired electrical coupling at Purkinje-myocardial junction-> re-entry
- HF=↓ $I_{to}$; ↓ $I_{K1}$; slower inactivation of $I_{Ca,L}$ in Purkinje and myocardium

# Recurrent VF Storms Acute Phase MI



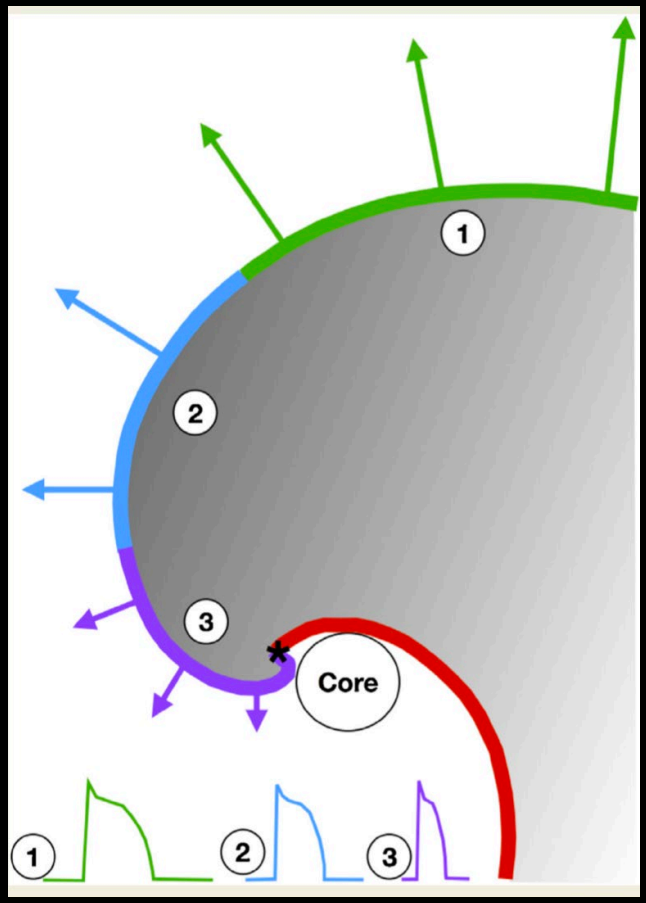



Damaged Purkinje Fibers
Ablation results in freedom from VF storm

Marrouche JACC 2004

# Transition phase VF

- Incompletely understood
- Triggering PVC → wavefront propagating through heterogenous areas of myocardium →wavebreak → functional re-entry (rotors)
- Early VF =large coherent wavefronts with intercalated disorganised wavelets with a limited number of epicardial drivers
- Rotors anchored to scar borders, anatomical/electrical discontinuities




Anderson, Kumar, Lee HLC 2019

# Maintenance phase VF

- Multiple wavelet vs. mother rotor hypothesis
- Multiple wavelet

  - multiple unstable circulating wavelets create self-sus- taining spiral wave re-entry.

- Mother rotor

  -mother rotor' is active, the ventricle is activated at high frequency, promoting wavebreaks and new driver formation perpetuating VF

- Likely that

  -Early phase VF-sustained due to multiple wavelets and rotors (mother rotor re-entry)

  -Long duration VF - maintained by focal Purkinje activity

# VF mapping

- Mechanism identifies: rotors and multiple wavelets, focal breakthrough RV), figure of eight re-entry
- VF maintained by limited number of drivers that often interacted.
- The type and the spatiotemporal behaviour of the different drivers:
  - reproducible between different VF episodes in the same patient.
  - varied considerably by underlying etiology
  - varied between patients with the same underlying etiology
- The limited number of drivers maintaining early VF and their reproducibility made them a target for ablation.

# VF in SHD

- VF rotors can interact and become stabilised and ‘anchored’ to scar
- VF in ICM triggered by damaged Purkinje fibers at scar border zone targeted for ablation[1]
- NICM – also at scar border zone but also at posterlateral LV close to MA[2]
- Empiric substrate ablation may be useful in ↓VF recurrences[3]

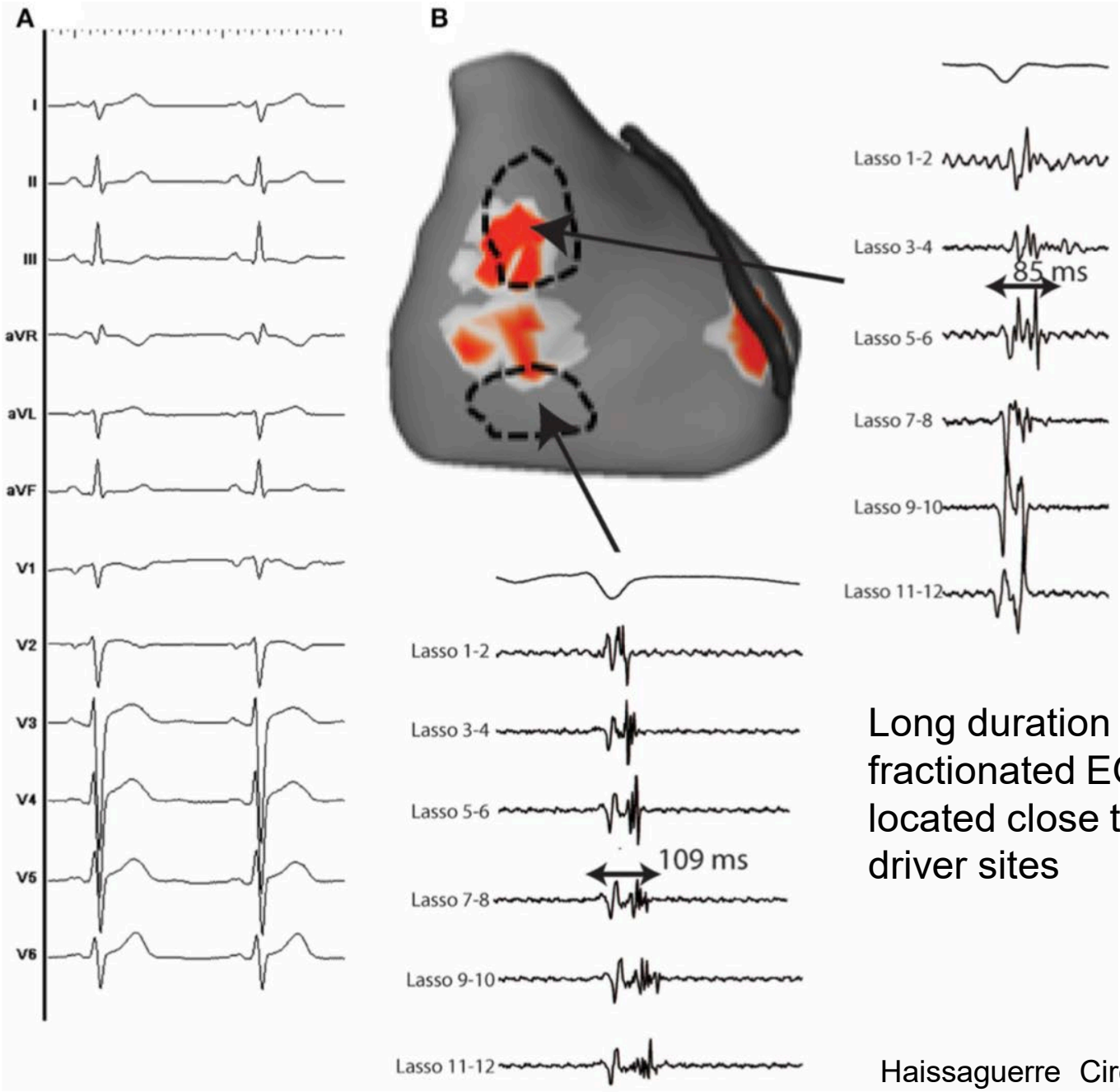
[1]Marrouche JACC 2002; [2]Sinha PACE 2009; [3]Nakamura HR 2019

# Substrate in IVF

- 24 IVF survivors; ECGi to map drivers of VF; high density endo epi biventricular mapping
- 19 VF episodes analysed (3 spont, 16 induced); mean 28 cycles during initial 5 s
- Mean 2.8 activities recorded per cycle (re-entrant in 87% and focal in 13%).
- Abnormal EGMs identified in 63% (confluent pattern over a limited surface area 5%)
- Predominantly epicardial

  -RVOT 11; LV 1; RV+LV 3
- Localized substrate co-located with the driver regions in 76%
- 7/9 pts without structural alterations had Purkinje triggers

Haissaguerre CircEP 2018




Long duration fractionated EGMs located close to VF driver sites

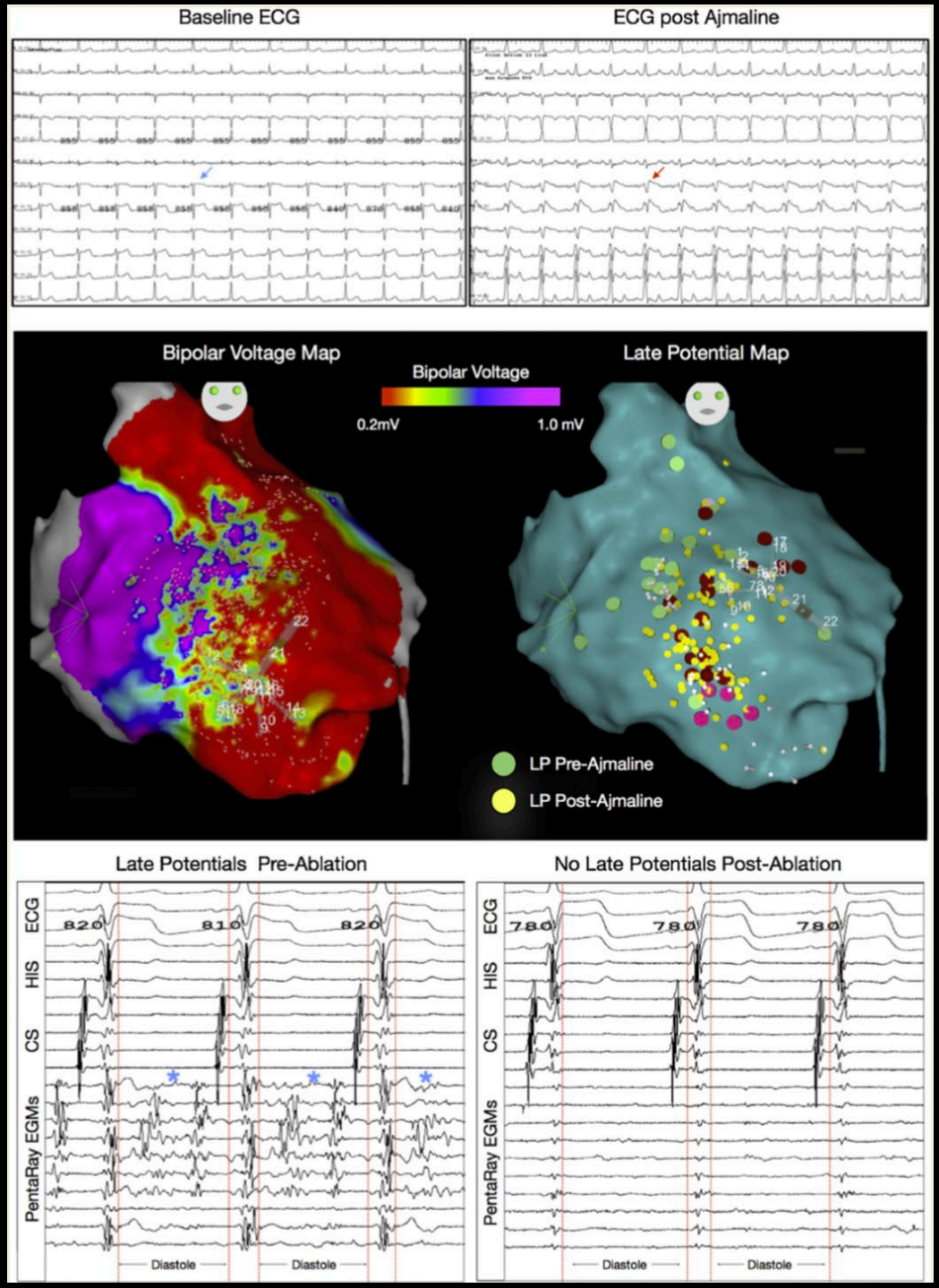
Haissaguerre CircEP 2018

# VF associated with ER

- ER patients exhibit 2 phenotypes
- Group 1 late depolarization abnormalities
  - -1A-concomitant BrS
  - -1B-no BrS
  - Late depolarisation areas co-located with VF drivers
    - Anterior RVOT/RV epi; inf RV major substrates
- Group 2: those without late depolarization abnormalities
  - -No ‘substrate’; Purkinje drivers
- Ablation targeting these areas 91% freedom from VF

Nademanee Circulation 2019

Group 1A:
concomitant BrS
(late depol)

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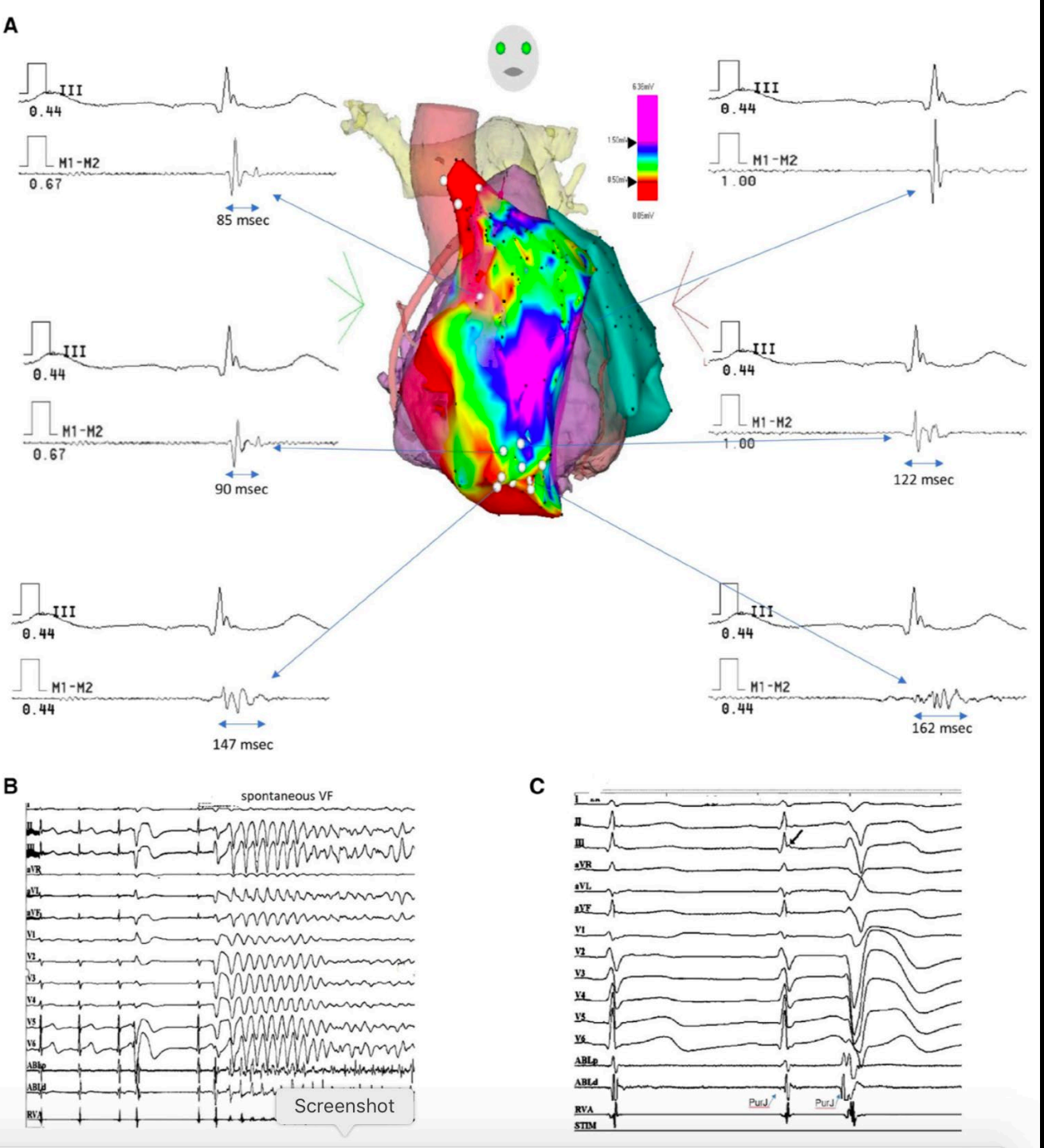
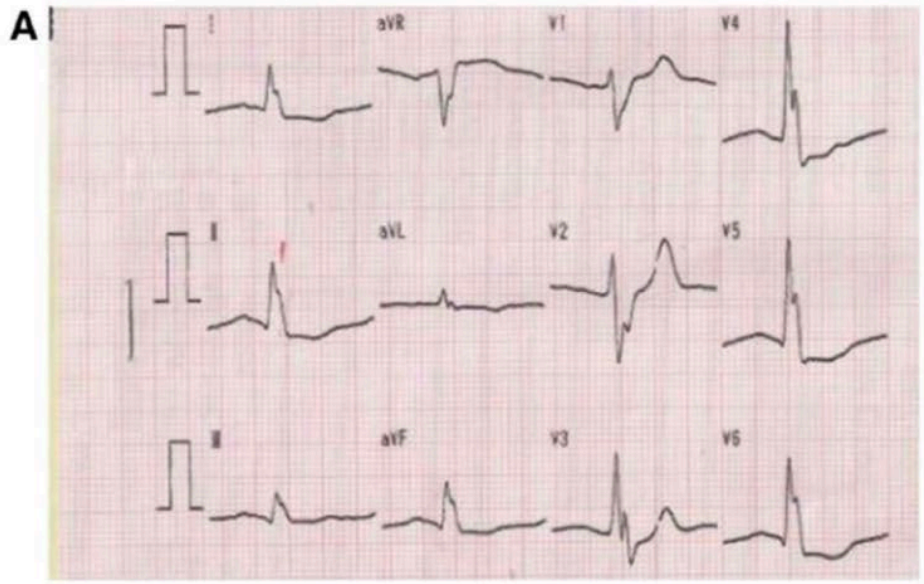
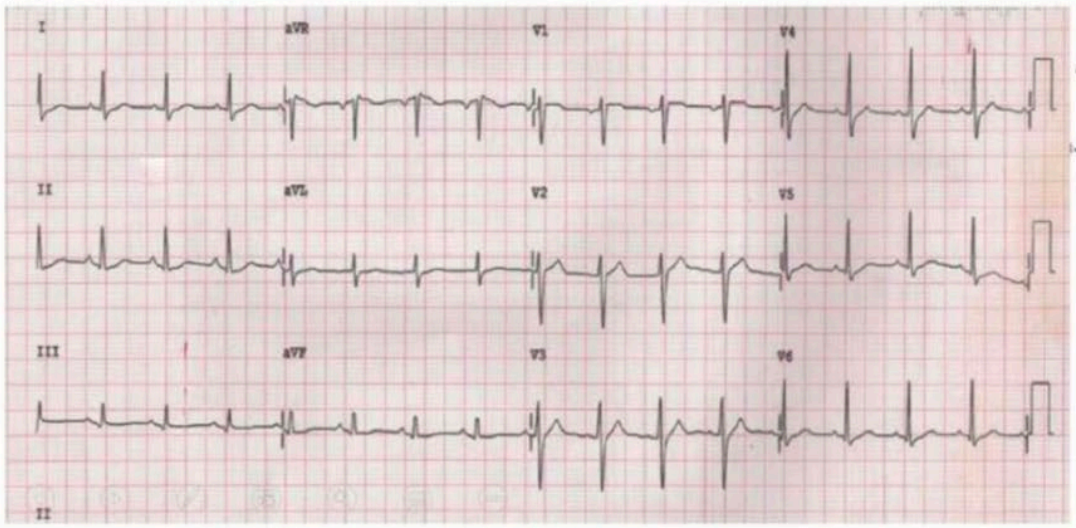
Kumar, Lee
HLC 2019

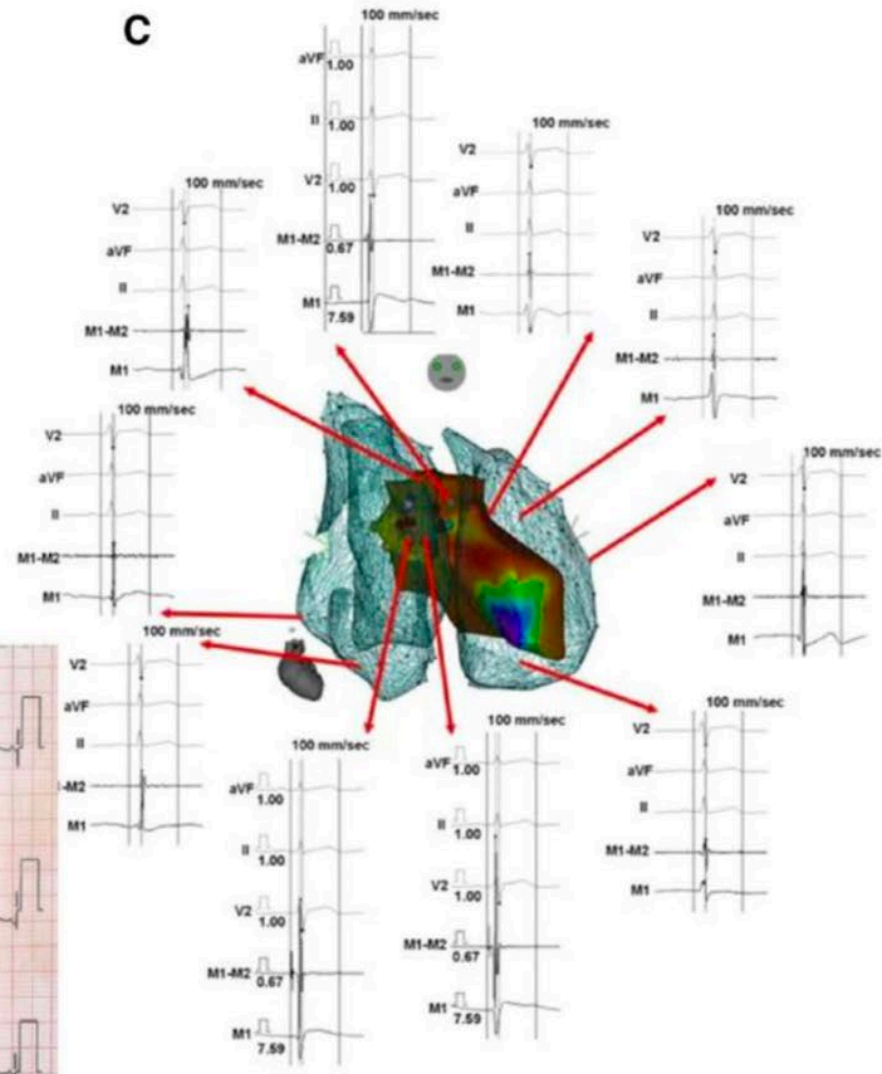
Group 1B: no BrS (late depol)



# Group 2: No late depol



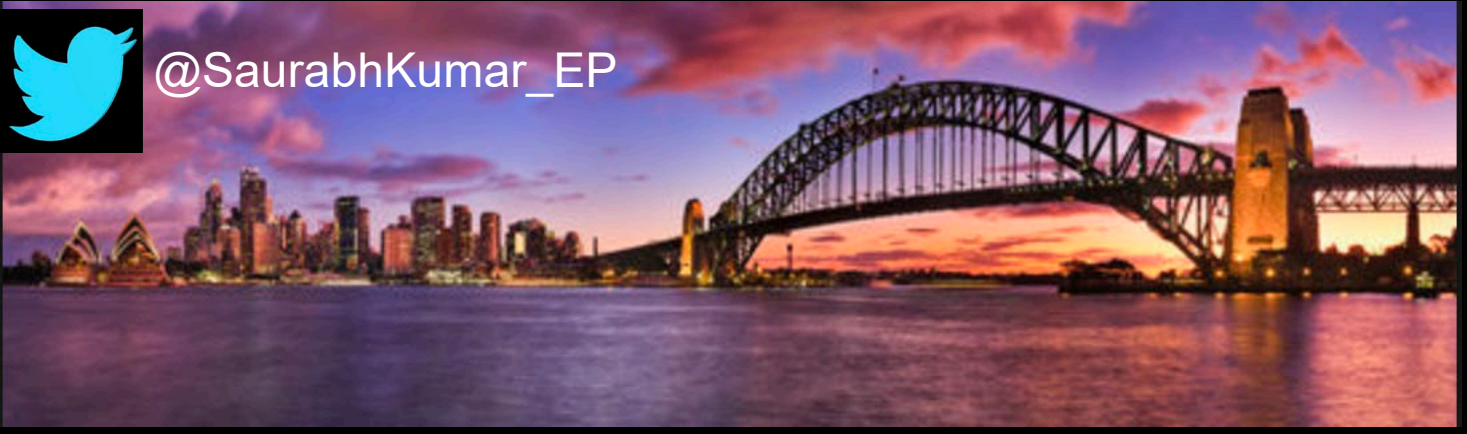
# Conclusions

- Catheter ablation important Rx for VF (preventing VF recurrences)
- All 4 phases of VF (initiation, transition, maintanece, evolution) serve as therapeutic targets for ablation
- Initiating PVCs (Purkinje) main target vast majority of VF
- Drivers maintaining VF emerging targets using ECGi
- Specific targets for VF exist according to disease type

  -SHD (Purkinje, scar)

  -ER (type 1A BrS; 1B RV; type 2 Purkinje)

  -IVF →concealed structural abn collocated next to VF drivers, amenable to ablation

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