

# Dual AV Nodal Non Re-entrant Tachycardia

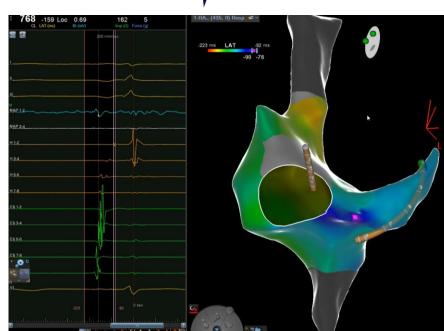
"Double Fire Tachycardia"

# **Mitchell Cowan**

(BSC ExSc, IBHRE CEPS, IBHRE CEPS, GDip CEPIA)

Cardiac Physiology in Practice,
Australia





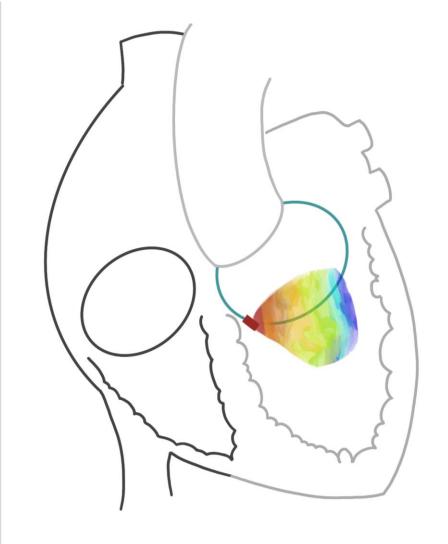


### **Financial disclosures:**

- Receives financial compensation from Cardiac Physiology in Practice EP & ECG education for healthcare professionals (current)
- Receives financial compensation for employment with Biosense Webster (current)
- Received financial compensation from Boston Scientific to produce Electrophysiology education (previous)
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### Non-financial disclosures

- Serves as Co-author for CONDUCT TAVI trial sponsored by Biotronik (current)
- Serves on Allied Health Board for IBHRE Allied Health CEPS writing committee (current)
- Served on Professional Standards Committee for Professionals in Cardiac Sciences Australia (previous)





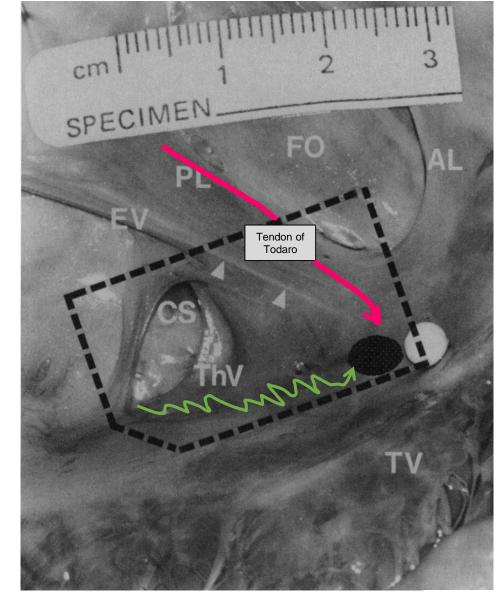
# Inferior Extension (Slow Pathway)

# **Anatomy**

• Inferior/posterior extension of the compact AV node, with a trajectory towards the lip of the CS ostium, posterior to the tricuspid annulus & valve apparatus.

## **Slow conduction velocity due to:**

- 1. Paucity of large Gap junctions (connexins 40/43)
- 2. Fibre disruption

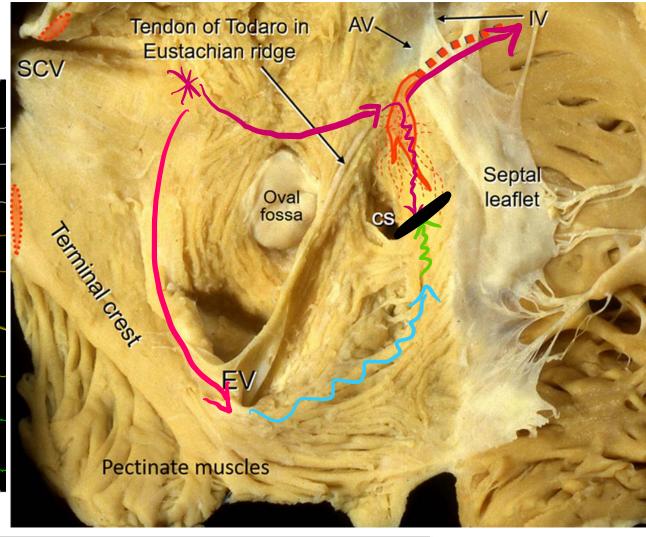




# CARDIAC PHYSIOLOGY

# Sinus Rhythm – fast pathway conduction



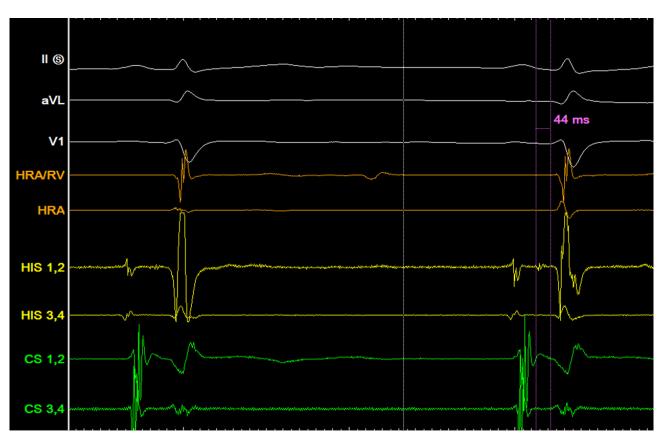


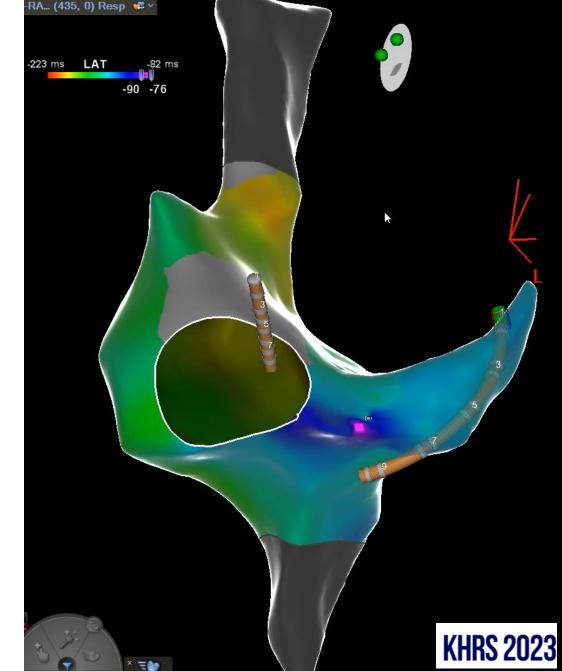
- Sinus rhythm results in antegrade conduction down inferior & superior extensions of AV node in patients with DAVNP.
- Conduction down "fast pathway" dictates PR interval & AH time.
- Wavefront collision occurs in slow pathway (retrograde from the fast pathway & antegrade from slow pathway)





Sinus Rhythm – fast pathway conduction







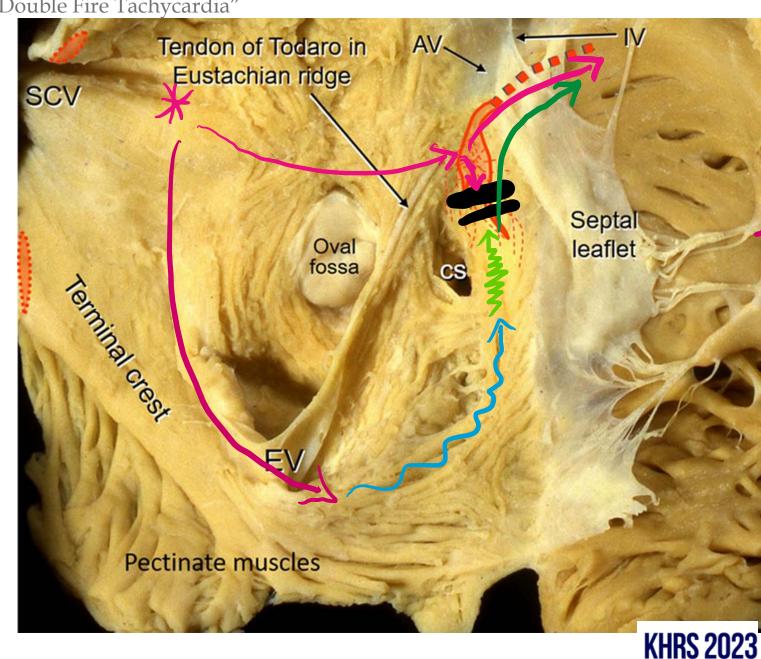
# DAVN Non-Reentrant Tachycardia



"Double Fire Tachycardia"

- 1. Sinus depolarisation results in depolarisation of the fast pathway with impartial retrograde penetrance of the slow pathway.
- Impartial retrograde activation may be due to anisotropic retrograde SP block, or the extinguishing of wavefront conduction due to acute wavefront geometry.
- 2. Antegrade Slow Pathway conduction velocity is sufficiently slow to facilitate AV nodal repolarisation and conduct an additional impulse to the ventricles.
- 3. Results in 1:2 A:V conduction.

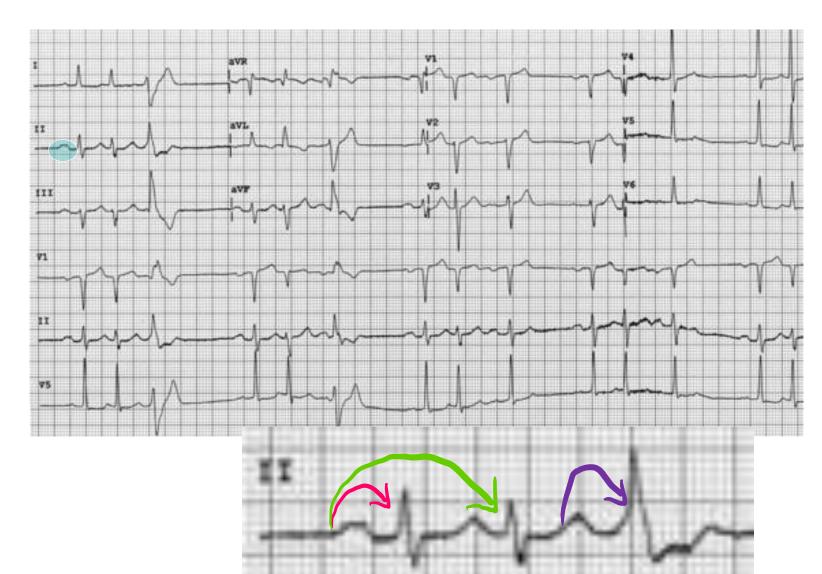
Nagarajan VD, Ho SY, Ernst S. Anatomical Considerations for His Bundle Pacing. Circ Arrhythm Electrophysiol. 2019 Jul;12(7):e006897. 🔐 🖟 u D Denes P Dhingra R Pietras R Rosen K. New manifestations of dual Anodal pathways. Eur J Cardiol 1975;2:459-66.



# CARDIAC PHYSIOLOGY IN PRACTICE

### **ECG** Characteristics

- 1. Is not always 1:2 A:V!! May appear irregular at first glance, perhaps suggesting atrial fibrillation.
- 2. Closer inspection will demonstrate clear P waves with some degree of QRS regularity present. "Couplets of QRS complexes"
- 3. QRS may be narrow or aberrantly conducted
- 4. May appear as frequent PAC's, PVC's or NSVT

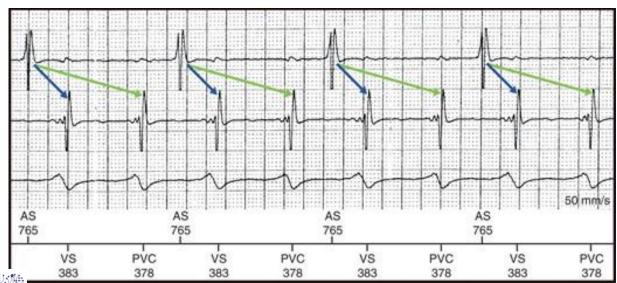


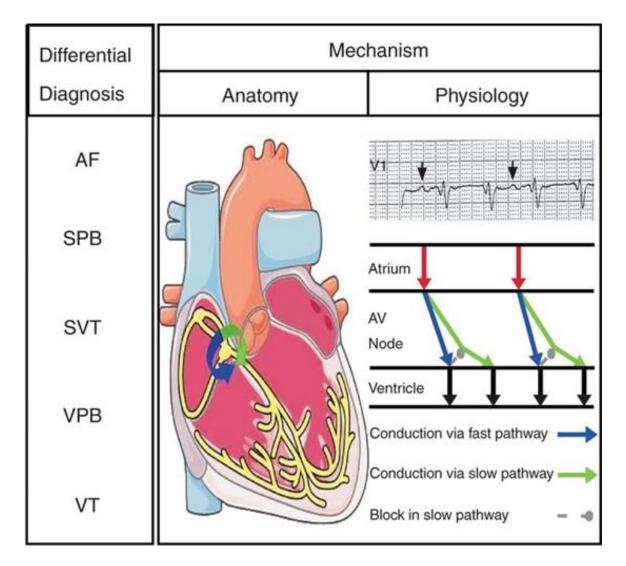


## Clinical Presentation



- 1. Similar Male: Female ratio with age between 16-84yrs
- 2. Structurally normal hearts (no evidence that structural abnormalities pre-dispose patients to DAVNNRT).
- 3. Symptoms include palpitations, dyspnoea, fatigue, dizziness & Tachycardia mediated cardiomyopathy





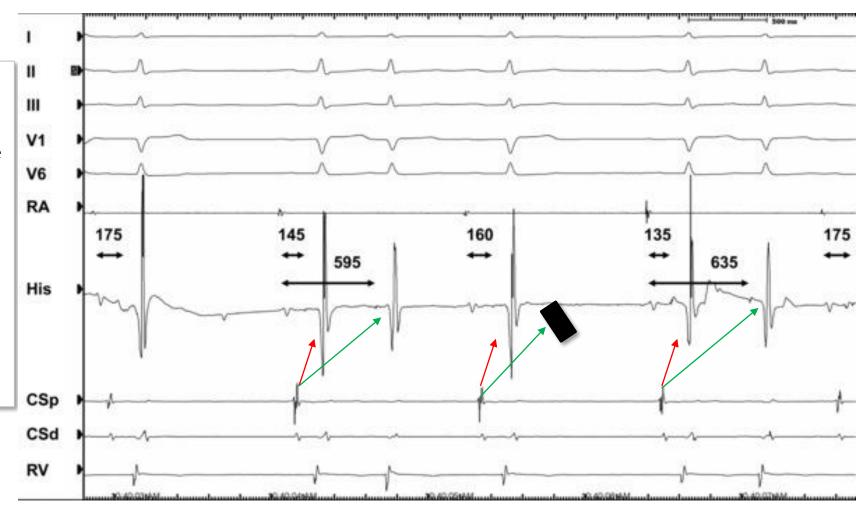
- Nagarajan VD, Ho SY, Ernst S. Anatomical Considerations for His Bundle Pacing. Circ Arrhythm Electrophysiol. 2019 Jul;12(7):e006897.
- Wu D Denes P Dhingra R Pietras R Rosen K. New manifestations of dual A-V nodal pathways. Eur I Cardiol 1975;2:459–66.
- Christiane Peiker et al. Dual atrioventricular nodal non-re-entrant tachycar KHRS 2 Volume 18, Issue 3, March 2016, Pages 332–339,





# **EP Diagnosis of DANNRT**

- 1. 1:2 Atrial conduction
- 2. Variation in AH interval may be present due to fluctuations in AV nodal decrementation.
- 3. HV interval should consistently >35ms indicating that the "extra" QRS complexes are originating from above the His bundle and therefore likely supraventricular in origin.





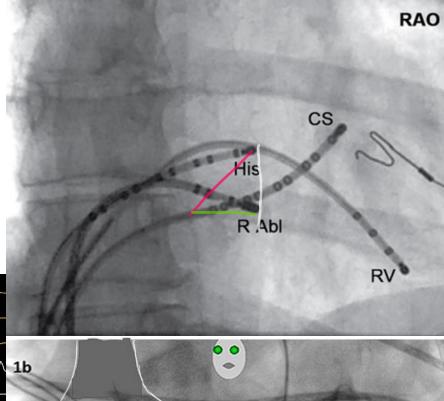


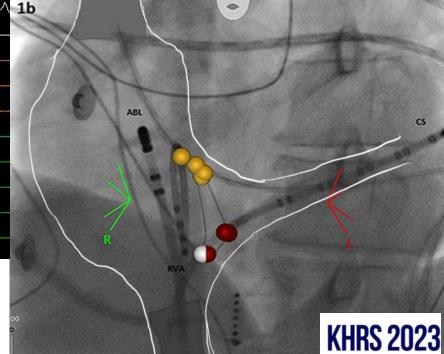
# Treatment & Endpoints

- 1. Slow Pathway ablation with abolition of DAVNP.
- 2. No inducible double fire response.







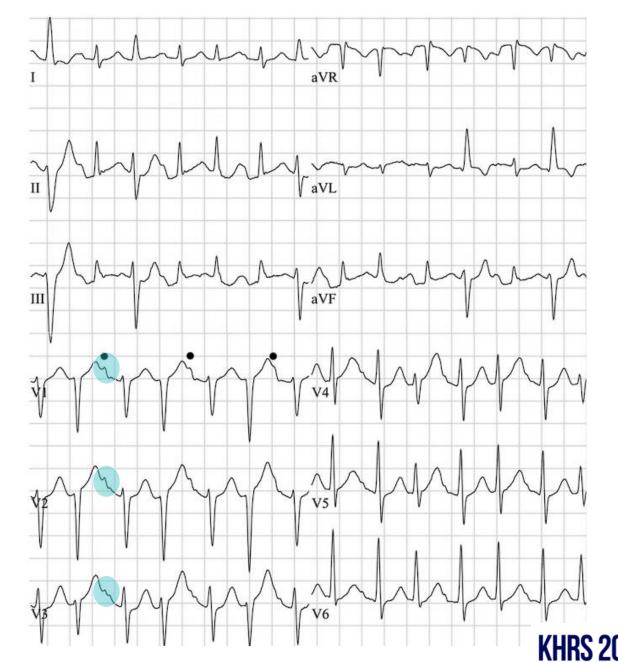






# Case Study: Presentation & ECG

- 1. 40-year-old male with Hx of infrequent palpitations treated with Beta Blockers due to presumed PACs or Junctional ectopic beats.
- 2. Palpitations became incessant & was referred to EP Lab.
- 3. No structural abnormalities.

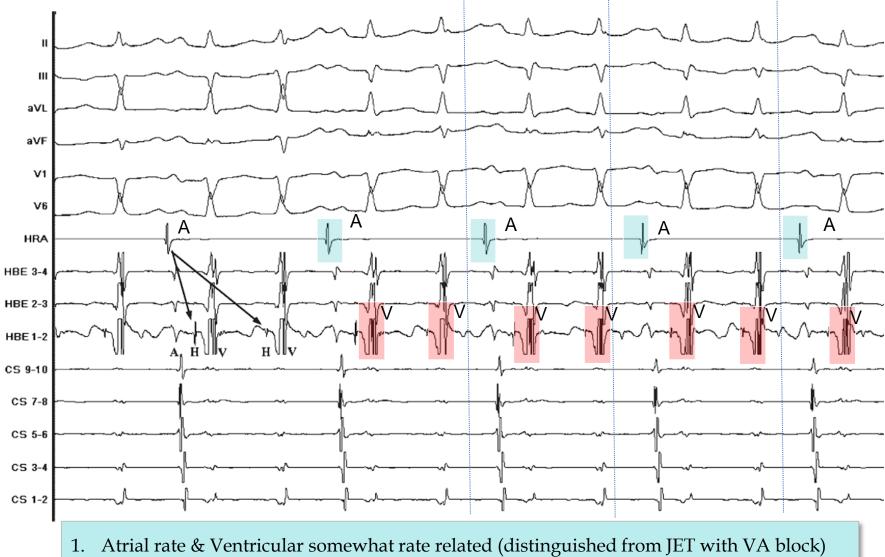






2. 1:2 AV ratio always.

# Case Study: EGM's

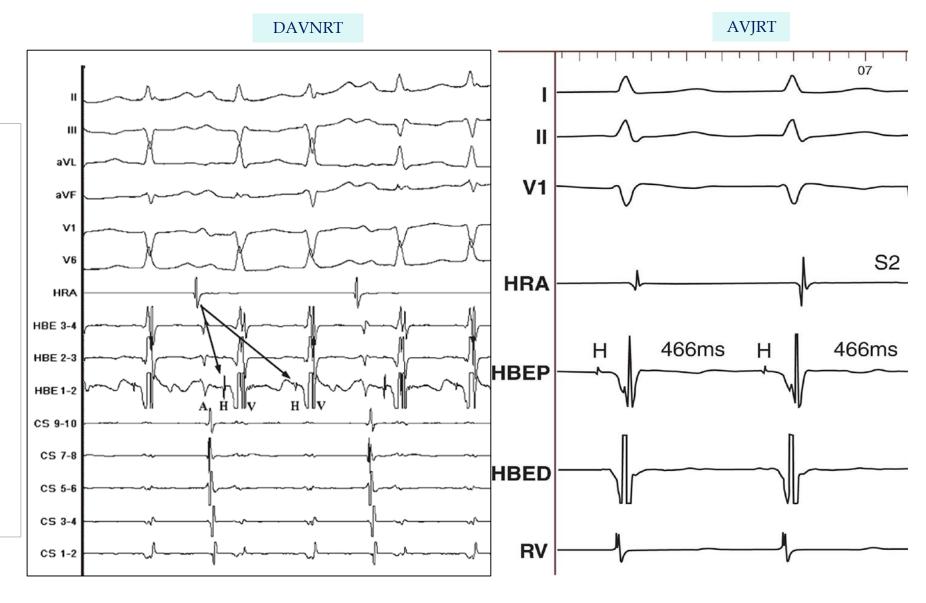






# Case Study: Differentiation from AVJRT

- 1. AHV sequence present with Physiologic AV interval.
- 2. VA interval consistently >70ms with long RP
- 3. P wave is upright II/III & of a sinus origin.
- 4. VAAV response to RV Overdrive Pacing (Tachycardia is NOT reentrant)







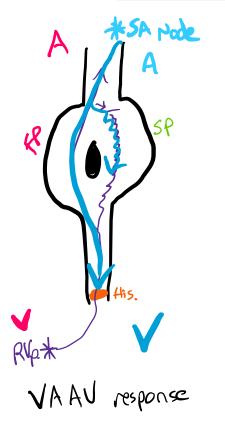
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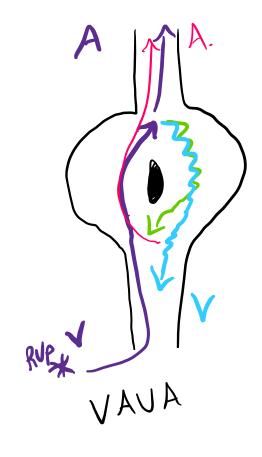
DAVNRT

AVJRT

 VAAV response to RV Overdrive Pacing

(Tachycardia is NOT re-entrant)







### 1. P wave morphology:

- Upright P waves in II/III/aVF with demonstratable AHV association → DAVNNRT
- Negative P waves in II/III/aVF  $\rightarrow$  JET

### 2. HV interval

- DAVNRT>35ms consistently.
- JET <35ms often

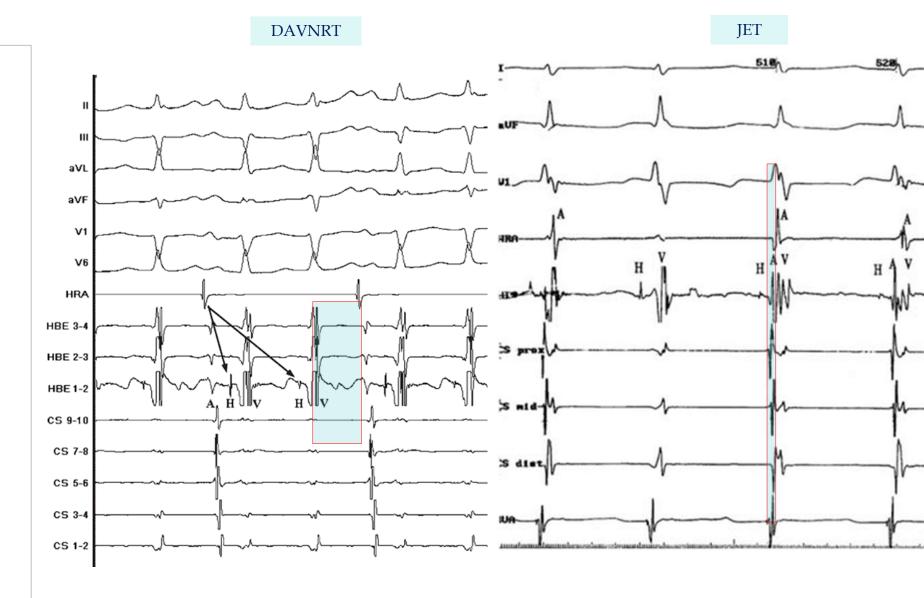
### 3. VA interval

- DAVNNRT >70ms
- JET <70ms

# 4. DAVNRT → AA interval predicts variation in VV interval.

- JET (HH changes precede AA changes)

# Case Study: Differentiation from JET







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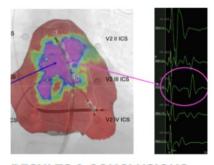


#### REVIEW

# BRUGADA ABLATION FOR RECURRENT VT/VF

#### **BRUGADA SYNDROME**

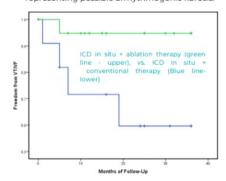
- Brugada 'Syndrome' represents a genetic disorder resulting in mutated sodium channels & 17% risk of sustained ventricular arrhythmias over a 7 year period (Brugada et al. 2015).
- Resulting heterogeneity in transmural repolarisation or depolarisation timing between the endo and epicardium, initiates phase 2 re-entry and ventricular fibrillation placing the patient at risk of sudden death & deleterious psychological outcomes from ICD shocks.
- Recent evidence suggests that depolarisation abnormalities may play a more prominent role than previously thought as EP studies have demonstrated areas of epicardial fibrosis in the RV outflow tract regions which may contribute to arrhythmogenesis.



# BRUGADA ABLATION: LATE BREAKING HRS RESULTS

Preliminary data is presented here from the unpublished research 'Epicardial Ablation In Brugada Syndrome To Prevent Sudden Death: Results From A Randomized Clinical Trial'. Presented by Giuseppe Ciconte in HRS 2022. It aims to recruit 150 patients.

- This is a randomised control trial comparing epicardial Brugada Ablation with ICD Vs ICD alone. The data presented here is based on 35 patients with at least 1 ICD shock or cardiac arrest
- Brugada ablation targeted areas of low voltage and fractionation made manifest with Ajmaline infusion (left image below).
- Procedural endpoint was homogenisation of anterior RVOT area with elimination of all abnormal activity & fragmented electrograms representing possible arrhythmogenic fibrosis.



#### **RESULTS & CONCLUSIONS**

- Ablation resulted in abolition of Type 1 Brugada ECG phenotype with no inducible Brugada pattern
  with ajmaline infusion. These results are in line with previous case reports and observational data sets.
- Ablation was also associated with significantly lower VT/VF recurrence compared to ICD alone (right image above). No report on Quinidine utilisation in each arm at this stage.
- Brugada ablation represents a paradigm shift in the ablation of a 'presumed channelopathy' towards a
  heart with subtle structural abnormalities.
- This and previous data, including Kumar's et al. 2022 systematic review of 388 patients with Brugada ablation, suggests that at least a subset of patients with diagnosed Brugada syndrome, may have evidence of RV fibrosis where ablation may alleviate some risk of VT/VF.
- Future randomised controlled data with larger patient cohorts are needed to confirm safety & efficacy
  of the technique compared to conventional therapies.

# CARDIAC Differentiation from NFV mediated Tachycardia with 2:1 VA conduction

### 1. His Synchronous RV pacing

- Will advance the immediate A and reset the V (cannot occur in AVJRT and JET if your RV stimulus is TRULY His Synchronous without the presence of an AP).
- AA advancement with VV reset is proof of AVRT.
- Even in VA block, may get VV "reset" without AA advancement.
- No advancement is a null result (not a negative proof).

### 2. Parahisian Pacing

• Often not useful in unmasking a Nodofascicular pathway as they tend to exhibit AV nodal behaviour with VA time increases when not capturing the His purkinjie system because they originate from the His purkinjie system. However, Nodoventricular pathways may be unmasked due to their insertion into ventricular myocardial tissue.

### 3. Upright P waves suggest DAVNNRT



# Differentiation from JET with 2:1 VA conduction



# I. P wave morphology:

- Upright P waves in II/III/aVF with demonstratable AHV association → DAVNNRT
- Negative P waves in II/III/aVF  $\rightarrow$  JET

### 2. <u>Driving electrogram?</u>

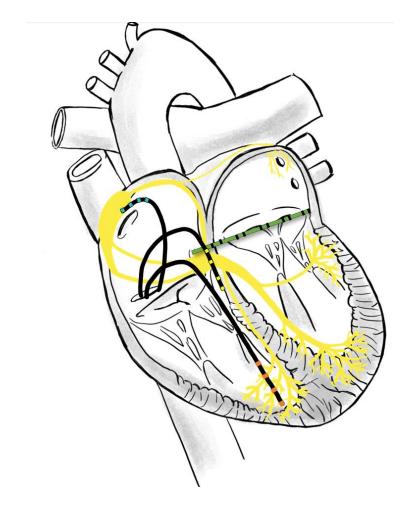
- Variation in AA intervals precede & predict variation in HH intervals → DAVNNRT
- Variation in HH intervals precede & predict variation in AA intervals → JET

### 3. VA time

- > 70ms with HRA earliest A EGM → DAVNNRT
- <70ms with His A earliest A EGM → JET

### 4. His Synchronous PAC

- Advancement of immediate His EGM related to slow pathway conduction indicates slow pathway presence.
- Slow pathway advancement of His EGM should not occur in JET due to wavefront collision.





# JET Differentiation

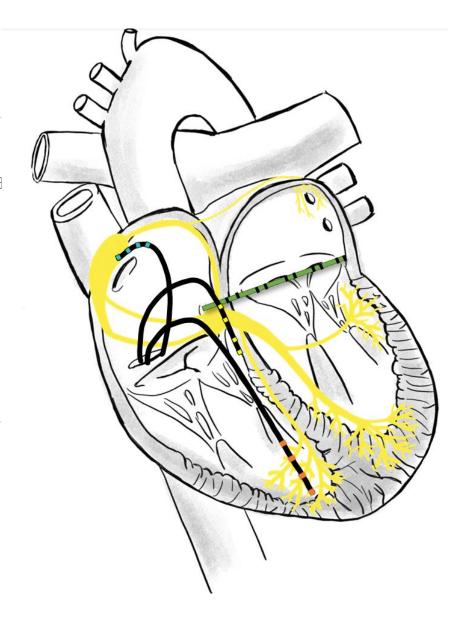


### Atrial overdrive pacing for an AHHA response.

- May work if you force 1:1 conduction during overdrive pacing. DANNRT will re-initiate with an AHAH response.
- JET will initiate with an AHHA response.

### Early PAC:

- Advancement of IMMEDIATE His EGM suggests fast pathway conduction without terminating tachycardia.
- Both JET and DANNRT will elicit this response. Not useful.



# Differentiating DAVNNRT from JET: His Synchronous PAC CARDIAC PHYSIOLOGY JET **DAVNNRT** AVN **AVN** His His ACL ACL

### <u>Introduction of PAC at the time of expected His or within 40ms after results in:</u>

- JET No advancement of subsequent His (Collision of antegrade & retrograde wavefronts)
- DANNRT: Any perturbation should exclude JET if PAC is truly His Synchronous?

Padanilam BJ, Manfredi JA, Steinberg LA, Olson JA, Fogel RI, Prystowsky EN. Differentiating junctional tachycardia and atrioventricular node re-entry tachycardia based on response to atrial extrastimulus pacing. J Am Coll Cardiol. 2008 Nov 18;52(21):1711-7.

Alasti M, Mirzaee S, Machado C, Healy S, Bittinger L, Adam D, Kotschet E, Krafchek J, Alison J. Junctional ectopic tachycardia (JET). J Arrhythm. 2020 Jul 27;36(5):837-844.

Differentiation from Typical AVJRT with UCPB

## 1. Ventricular overdrive pacing:

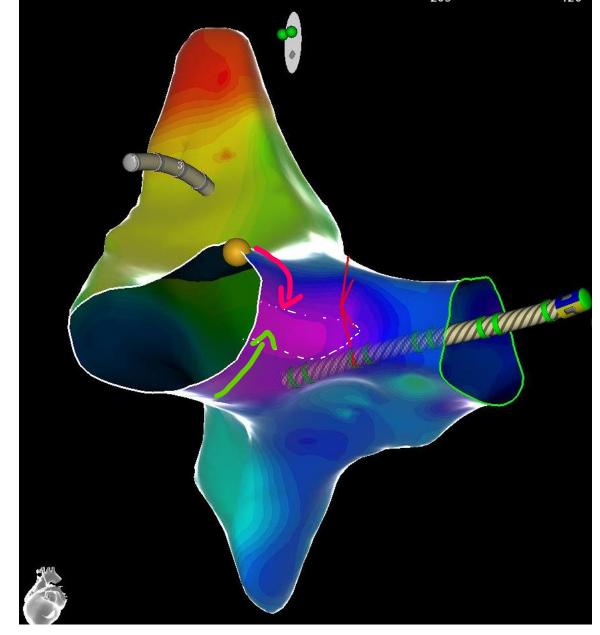
- AVJRT → VAVA response
- DANNRT  $\rightarrow$  VAAV response

### 2. <u>VA time <70ms</u>

- AVJRT <70ms
- DAVNNRT >70ms.

## 3. P wave morphology & earliest EGM

- Upright P waves with HRA earliest → DANNRT
- Negative P waves in II/III/aVF with His A leading → AVJRT



Differentiation from Atypical AVJRT with UCPB



## 1. Ventricular overdrive pacing:

- AVJRT  $\rightarrow$  VAVA response
- DANNRT → VAAV response

### 2. P wave morphology & earliest EGM

- Upright P waves with HRA earliest → DANNRT
- Negative P waves in II/III/aVF with His A leading → AVJRT

## 3. VA linking with Atrial overdrive pacing

- No VA linking suggestive of DANNRT
- VA linking suggestive of AVJRT

