Dr. Jasbir Sra, MD, FACC, FAHA, FHRS

No Conflict

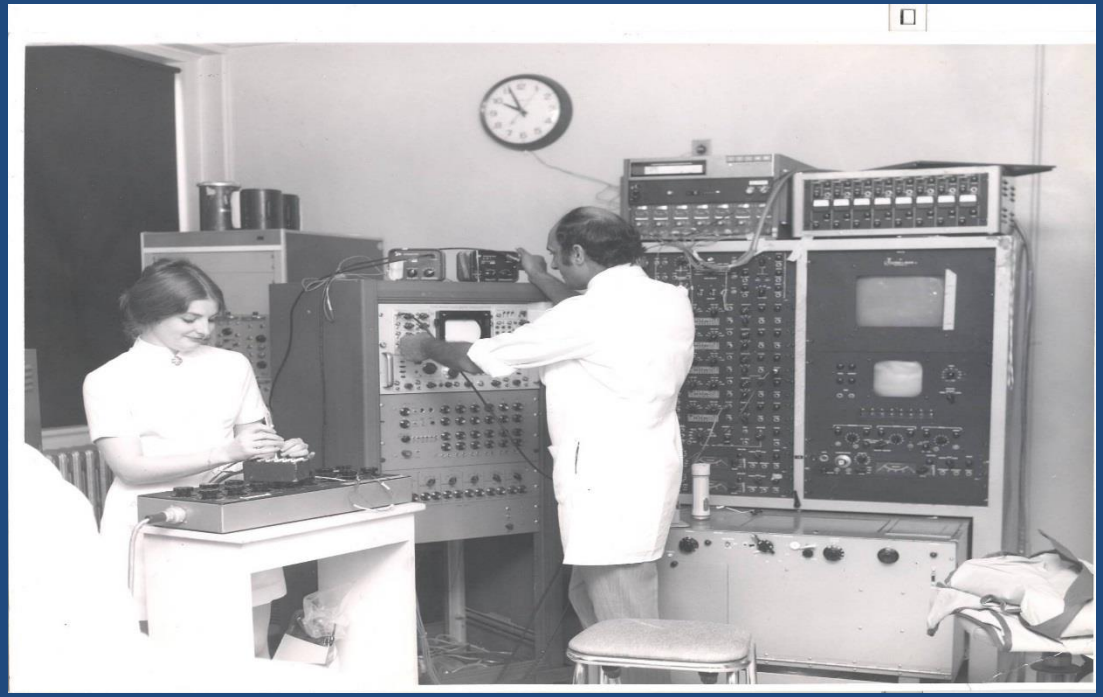
History of EP in Milwaukee

Dr. Masood Akhtar, MD

Dr. Donald Schmidt, MD

DR. Dudley Johnson, MD

History of EP in Milwaukee



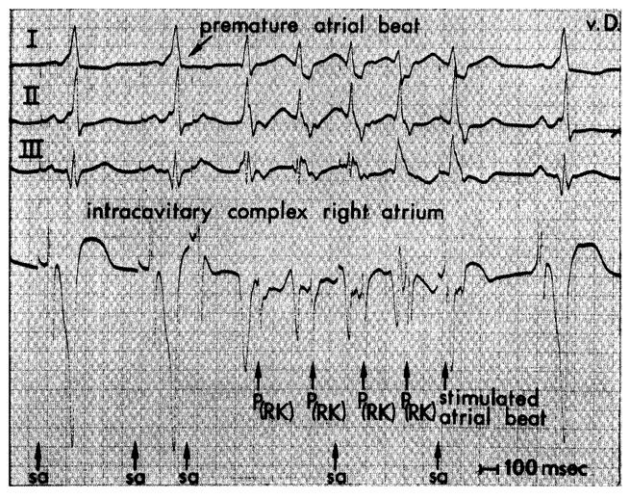
Mount Sinai-Now Aurora Sinai

The Role of Premature Beats in the Initiation and the Termination of Supraventricular Tachycardia in the Wolff-Parkinson-White Syndrome

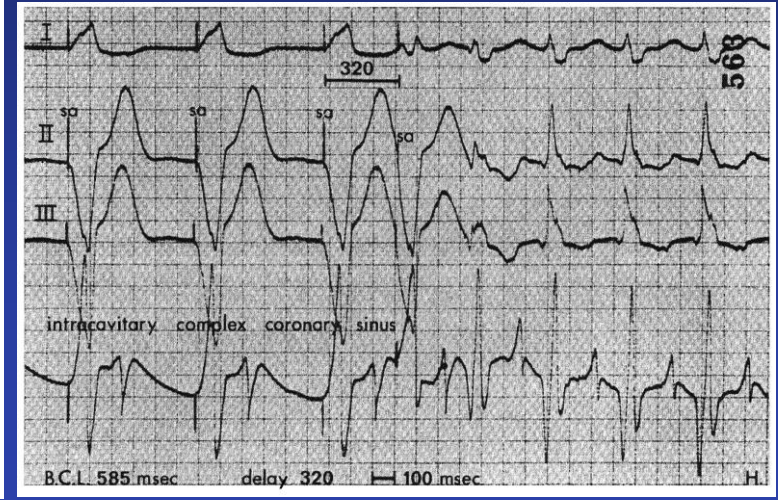
By D. DURRER, M.D., L. SCHOO, R. M. SCHUILENBURG, M.D., AND H. J. J. WELLENS, M.D.

Circ., Nov., 1967

Initiation and termination of supraventricular tachycardia during stimulation of right atrium

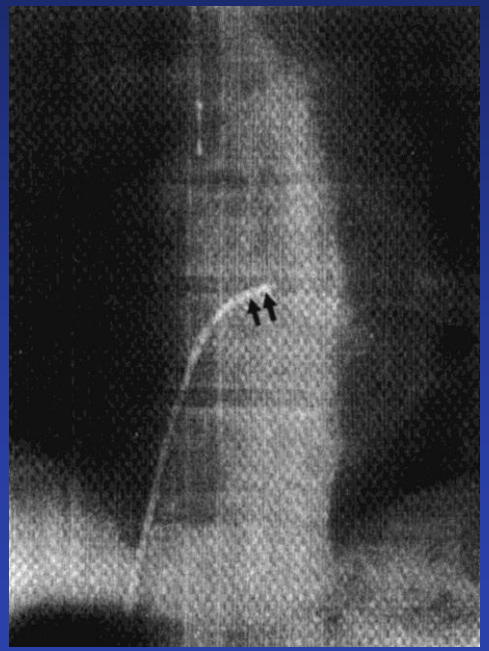


Regular driving of right ventricle and initiation of supraventricular tachycardia by induced right ventricular premature beat

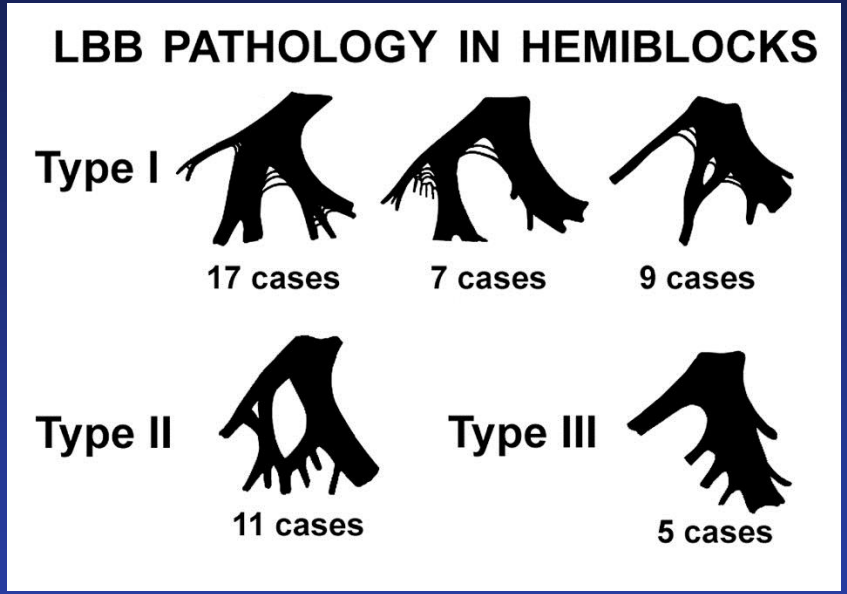


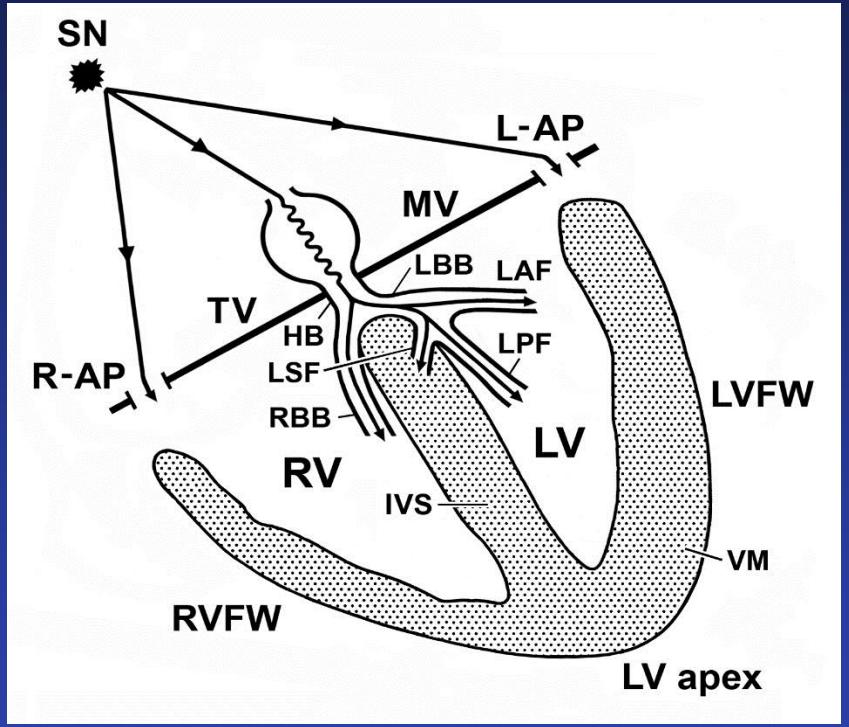
Catheter Technique for Recording His Bundle Activity in Man

By BENJAMIN J. SCHERLAG, PH.D., SUN H. LAU, M.D., RICHARD H. HELFANT, M.D., WALTER D. BERKOWITZ, M.D., EMANUEL STEIN, M.D., AND ANTHONY N. DAMATO, M.D.



LBB Pathology-49 Human Hearts





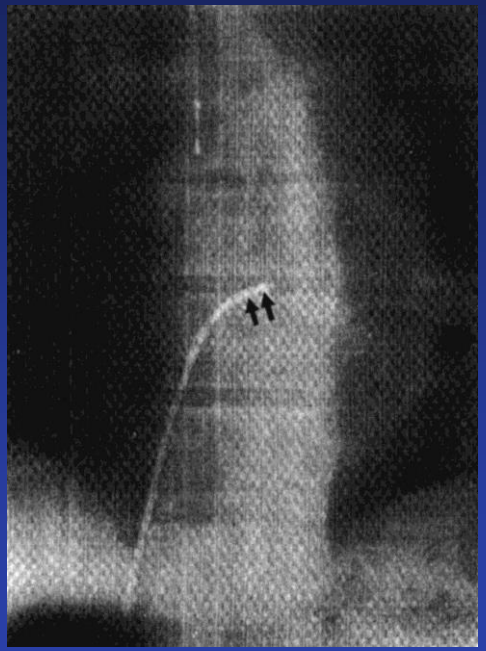
31 had a distinct 3rd division

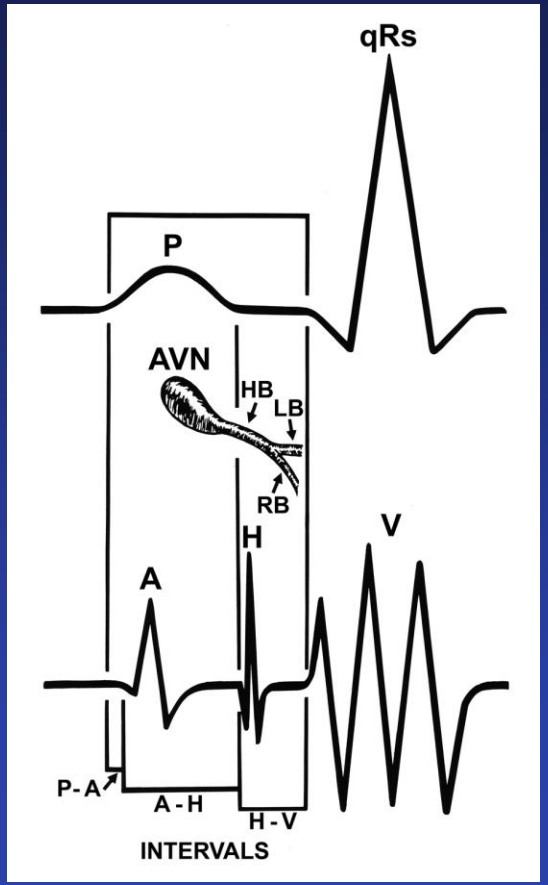
Kulbertus et al. in Wellens eta Editors, Springer, 1978

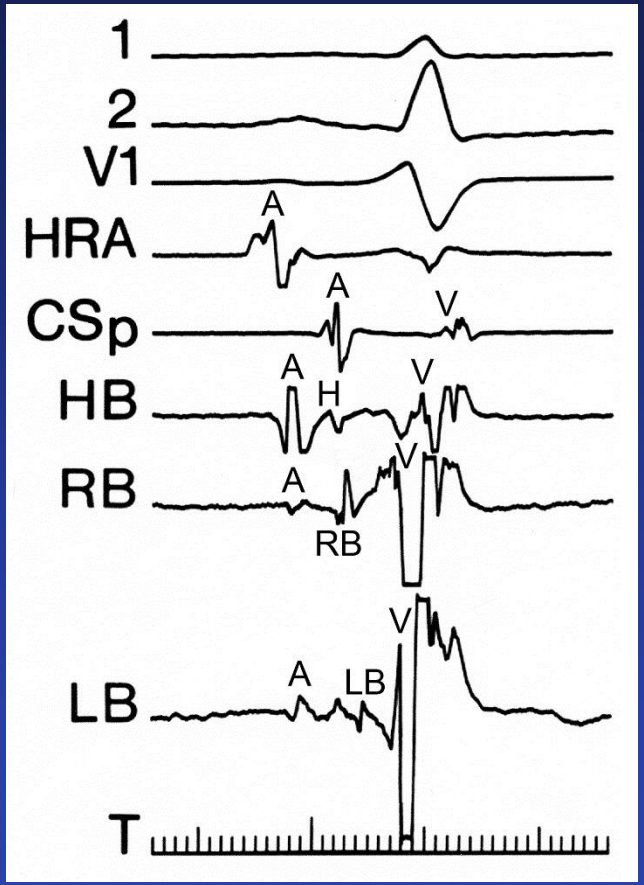
The following signatures are illustrated: 1. **Masood Akhtar**. Studies on physiology and reentry in the bundle branch system. 2. **Maurits A. Allessie**. Studies on atrial flutter. 3. **Agustin Castellanos**. Studies on parasystole. 4. **Philippe Coumel**. Arrhythmias on long-term electrocardiogram (ECG) monitoring. 5. **Jim L. Cox**. Localization and surgical excision of accessory pathways in the AV groove. 6. **Guy Fontaine**. Electrical fulguration of ventricular tachycardia. 7. **John J. Gallagher**. Electrical ablation of the bundle of His. 8. **Gerald Guiraudon**. Surgical treatment of ventricular tachycardia by encircling ventriculotomy. 9. **Michiel J. Janse**. Studies on ischemic arrhythmias in the pig heart. 10. **Mark E. Josephson**. Resection of the site of origin (of ventricular tachycardia). 11. **Richard Langendorf**. Reentry. 12. **William J. McKenna**. Arrhythmias and sudden death in hypertrophic cardiomyopathy. 13. **Michel Mirowski**. The implantable defibrillator. 14. **Robert J. Myerburg**. Studies on arrhythmias in the dog's heart. 15. **Peter J. Schwartz**. The long AT syndrome and torsade de pointes. 16. **Michael B. Simson**. Late potentials on the surface electrocardiogram (ECG). 17. **Harold C. Strauss**. Sinus node function. 18. **Albert L. Waldo**. Entrainment (continuous resetting of a reentrant circuit). 19. **Henrick J. J. Wellens**. Electrophysiologic studies in humans with multielectrode catheters. 20. **Andrew L. Wit**. Anisotropy of heart muscle in infarcted regions. 21. **Douglas P. Zipes**. The implantable cardioverter.

From Brugada, P. (1986) How some arrhythmologists should sign and why. *American Journal of Cardiology*, **58**, 381–2, with permission.

Retrograde & anterograde conduction in HPS

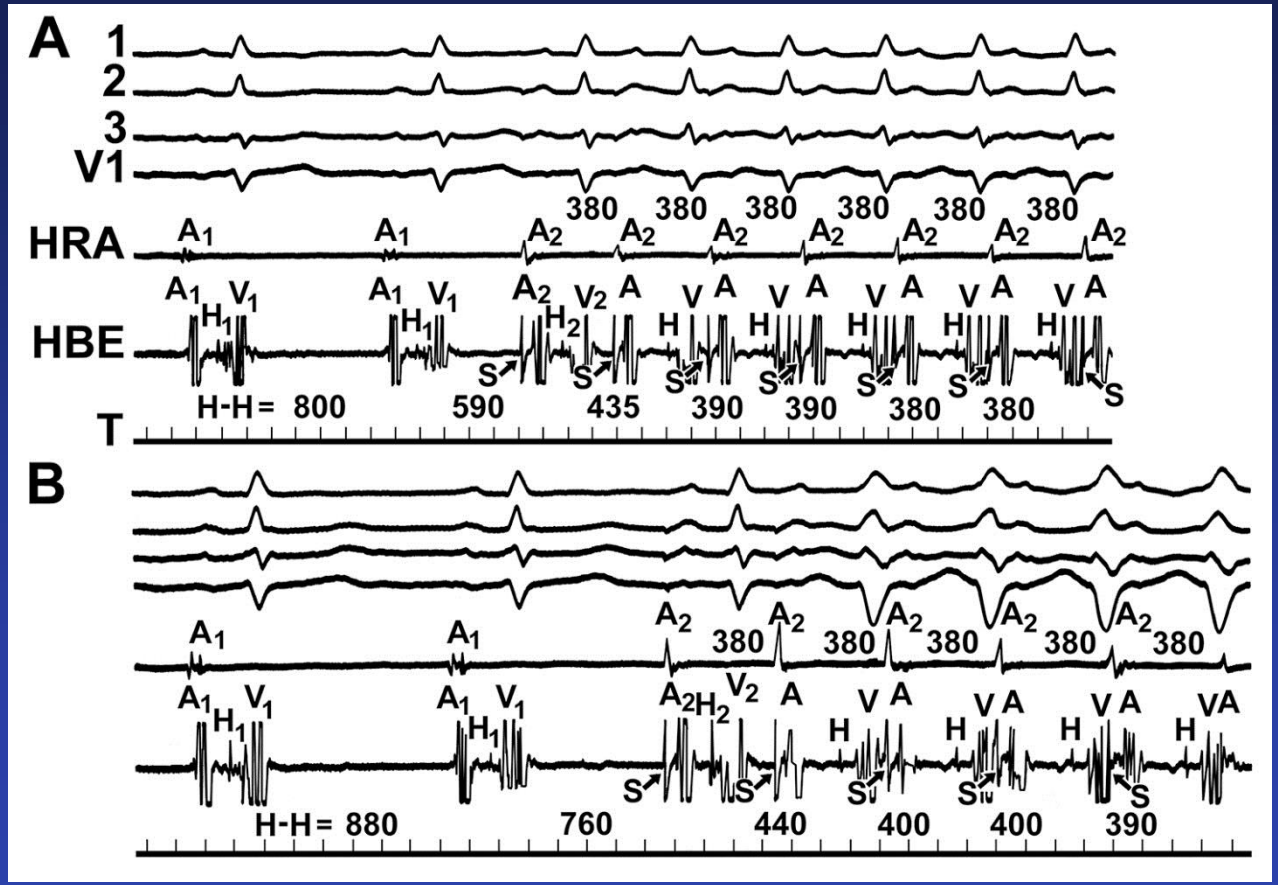






Normal His-Purkinje Physiology

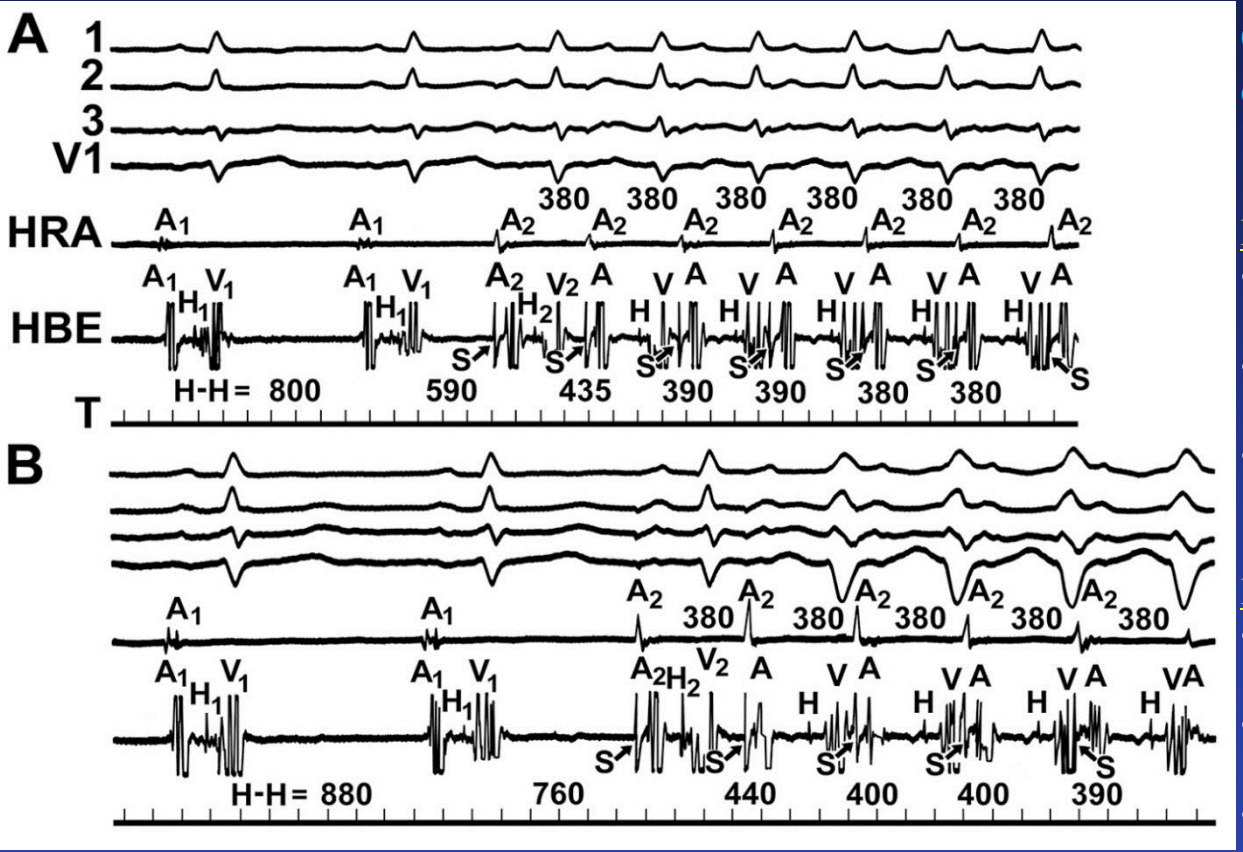
Response of His Purkinje System During Antegrade Impulse propagation



Q-why do we see LBBB in Panel B

Normal His-Purkinje Physiology

Response of His Purkinje System During Antegrade Impulse propagation



Key Factor is A1A2 to next A2A2 intervals

Q-why do we see LBBB in Panel B during random pacing

1. Panel A and B pacing at same CL

Panel A

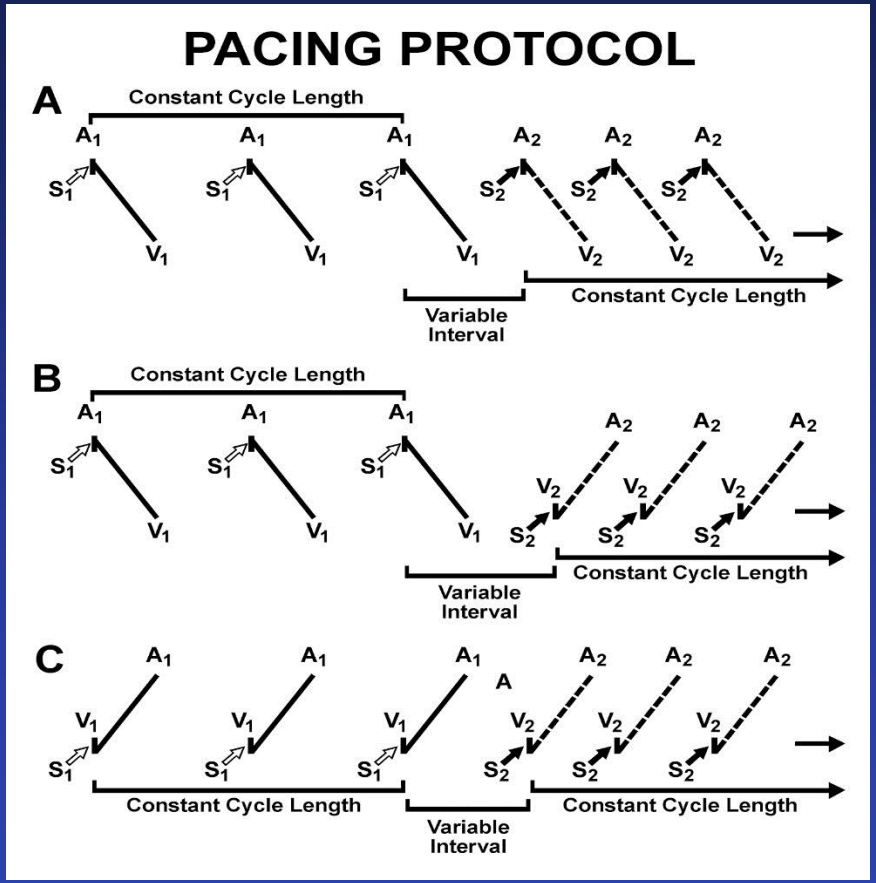
- Coupling interval (CI) of the of A1-A2 is 590 ms preceded by 800 ms
- A2-H2 prolongs and H2-H prolongs to 435 ms (73% of previous CL of 590 ms)
- Normal conduction occurs as His refractoriness is not achieved

Panel B

- A1-A2 is 760 ms-H2-H is 440 ms (53% of previous CL 760-440 ms)
- Relative shortening of H-H approaches refractoriness of LBB
- LBB continues due to retrograde concealed conduction or linking

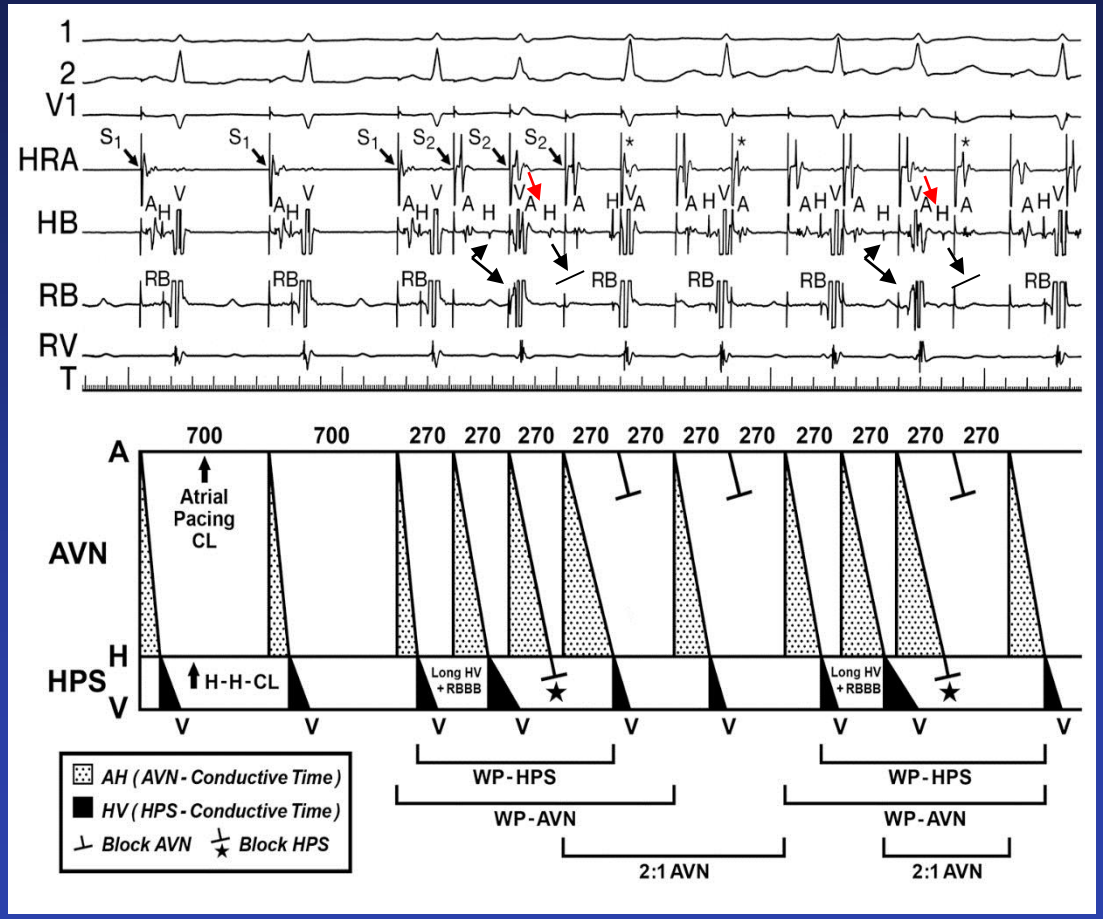
Study of His-Purkinje Physiology

Pacing protocols created to avoid random CL variation



Normal His-Purkinje Physiology

Response of His Purkinje System During Antegrade Impulse propagation



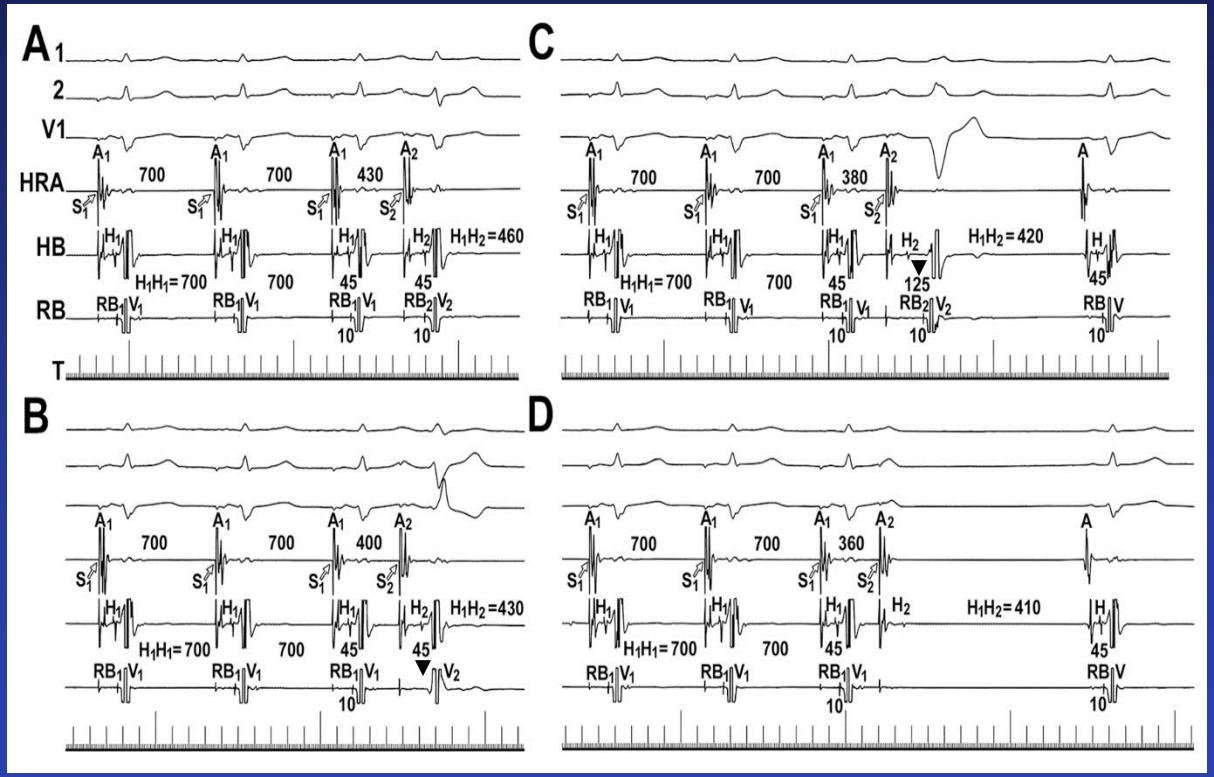
Sudden Change in atrial CL (700-270 ms) and HPS behavior

- S1-S2 same as the S2-S2 train
- The 2nd and 9th S2 blocks in HPS
- 4th, 6th and 10th block in the AV-Node
- Both HPS and Wenckebach cycle preceded by long H-H intervals First after S1-S2 second after AV block

Normal responses of the Heart: Akhtar et al, 1986;

Normal His-Purkinje Physiology

HPS Response to Single Atrial Extrastimulus



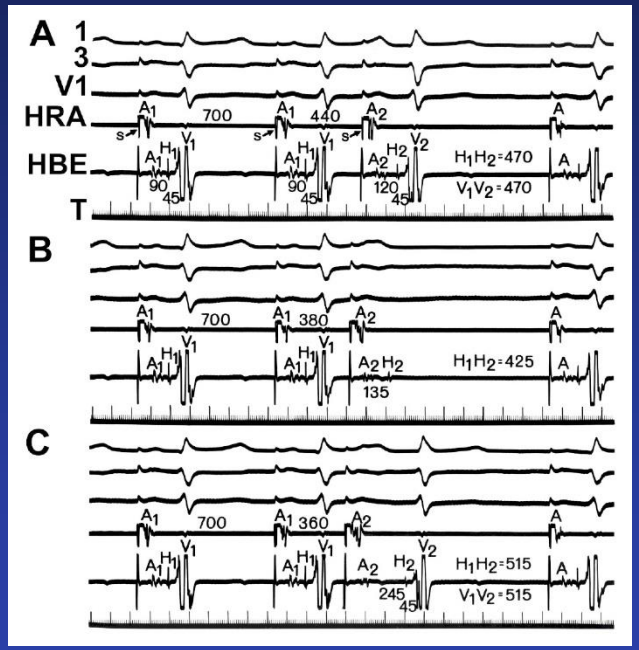
Basic SL 700 ms-A1-A2 coupling interval progressively shortened from 430 to 360 ms

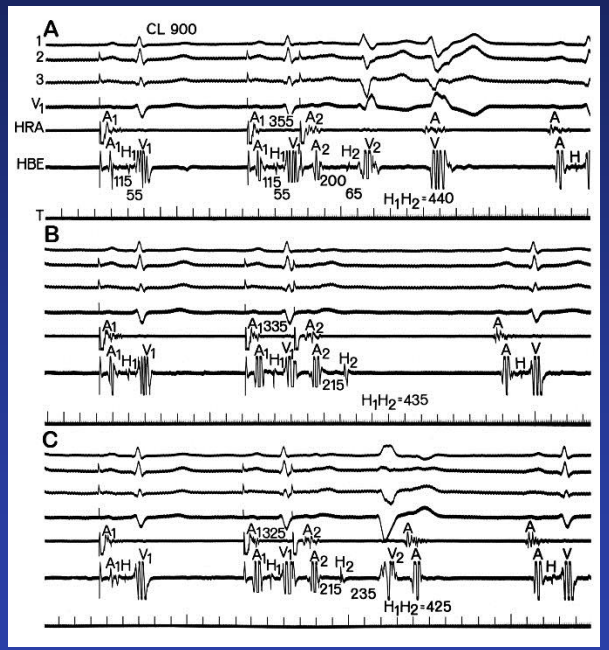
- A- Minor superior axis shift
- B-RBBB and LAFB
  - Block between H and RB
  - H_2V_2 remains normal as conduction in posterior fascicle is normal
- C-LBBB, H_2-V_2 prolongs to 125 ms
  -Conduction delay between H and RB
  - RB appears due to forward migration and disappearance of block due to delay in H-RB
- D- Infrahisian Block

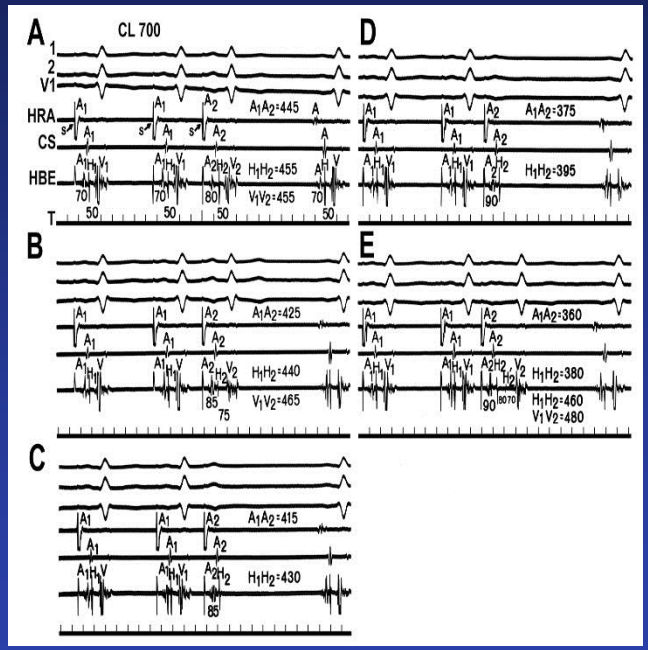
Site of conduction delay in HPS and block, Akhtar et al, 1980;circulation

Normal His-Purkinje Physiology

Q:-What causes conduction to reappear





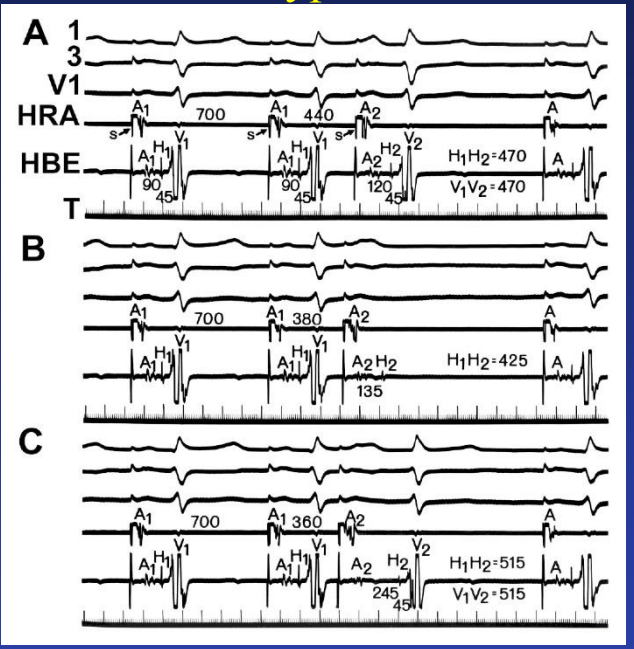


Akhtar M, Damato AN; Circulation 1974 . *Damato AN, Akhtar M, Ruskin J in Welllens HJ Lea and Febiger; 1974*

Normal His-Purkinje Physiology

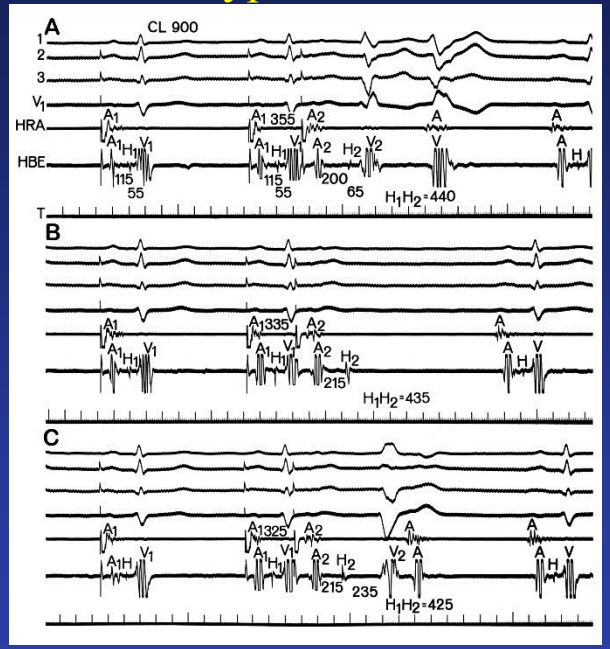
Gap Phenomenon

Type 1



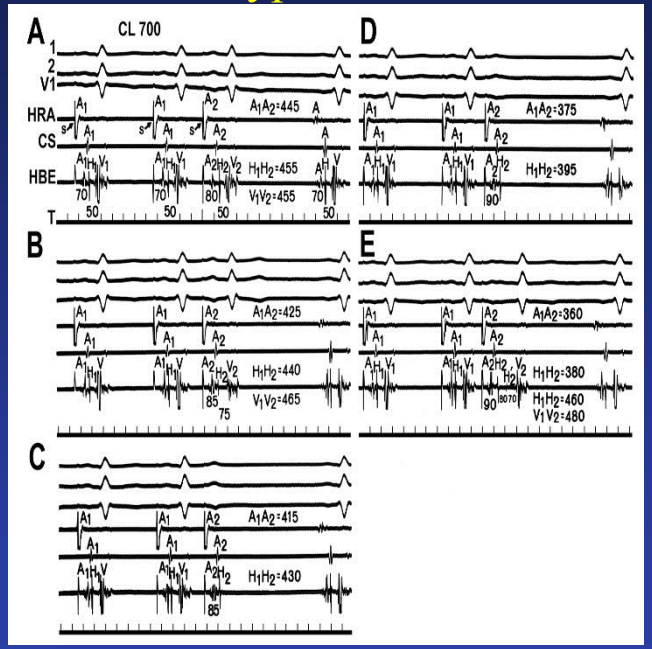
AV Nodal Delay

Type 2



Infra Hisian Delay

Type 3

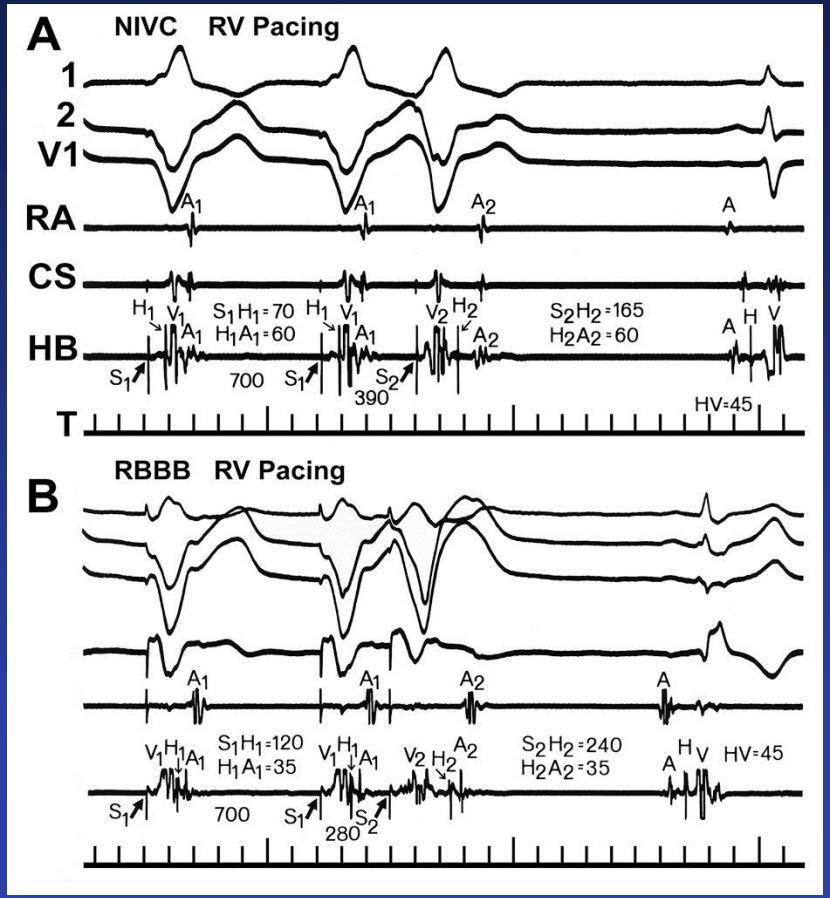


Intra Hisian Delay

RBBB disappears due to forward migration of the block
LBBB-But HV prolongs due to delay in RBB

Akhtar M, Damato AN; Circulation 1974 . *Damato AN, Akhtar M, Ruskin J in Welllens HJ Lea and Febiger; 1974*

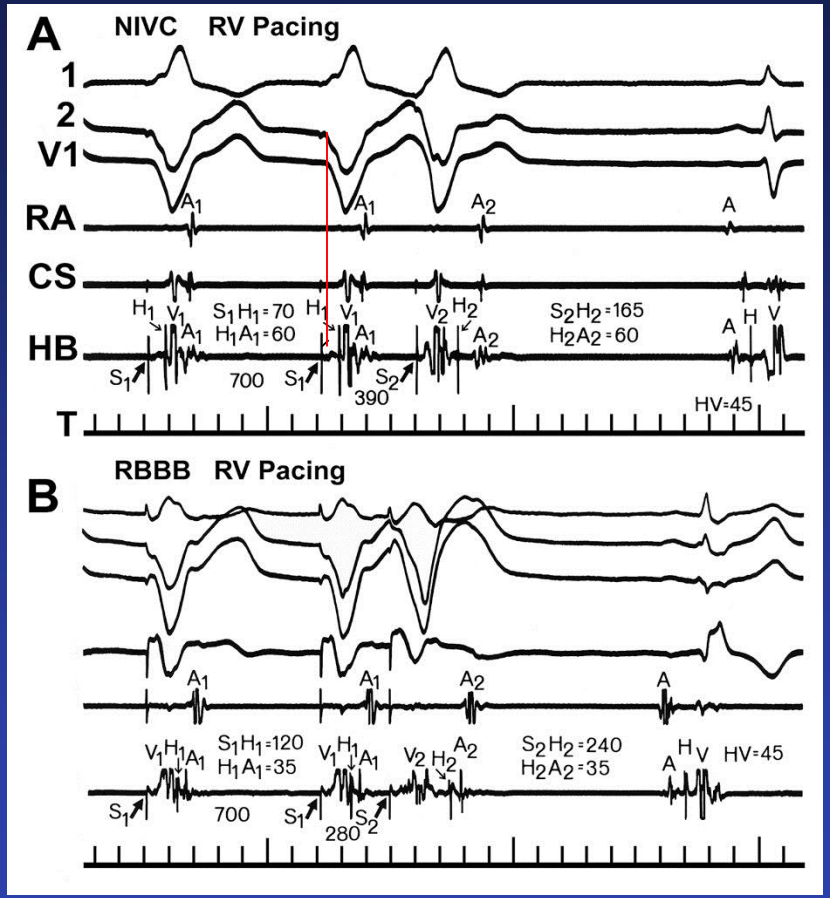
His-Purkinje During Retrograde Conduction



Q: Why is His showing up after V2

Akhtar M, Cardiac Electrophysiology Clinics

His-Purkinje During Retrograde Conduction



Akhtar M, Cardiac Electrophysiology Clinics

A and B are different patients

Panel A-NSR at baseline

- Retrograde His seen during S1-S1(rapid conduction and brief duration)
- But confirmed during S2
- During S2, V2 blocks in RB and conducts over LB- Transeptal delay brings the H2 out

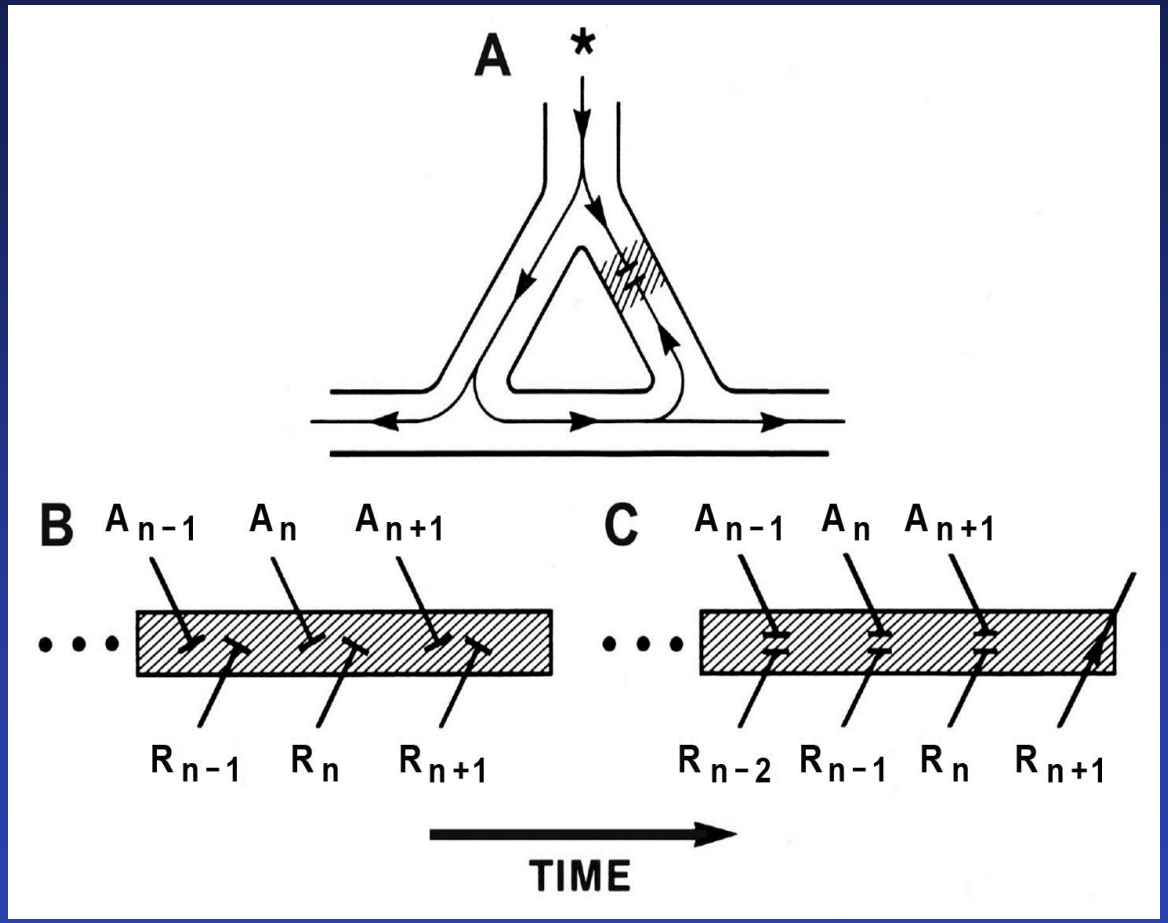
Panel B-RBB at baseline

- H1 farther out after V1 even during S1 due to RBBB
- After S2 H2 even farther out due to transeptal conduction but also delay in LB

S2-H2 -165 ms in A

S2-H2- 240 ms in B

Normal His-Purkinje Physiology-Linking



Linking Phenomenon

- A-Classic model of reentry
- B. Linking by interference

-impulse blocks but leaves an area of refractoriness which causes block of next beat and linking continues

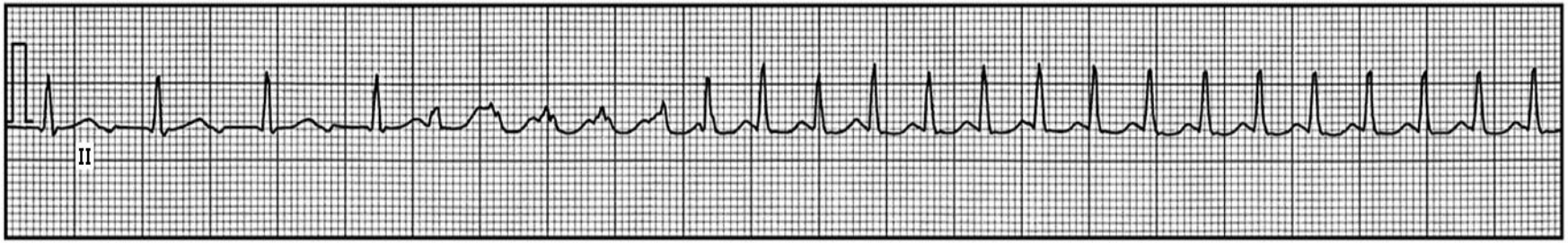
- C.-Linking by Collision

-Paced impulse enters excitable gap and conducts orthodromically to reset and collide antidromically with the previous beat

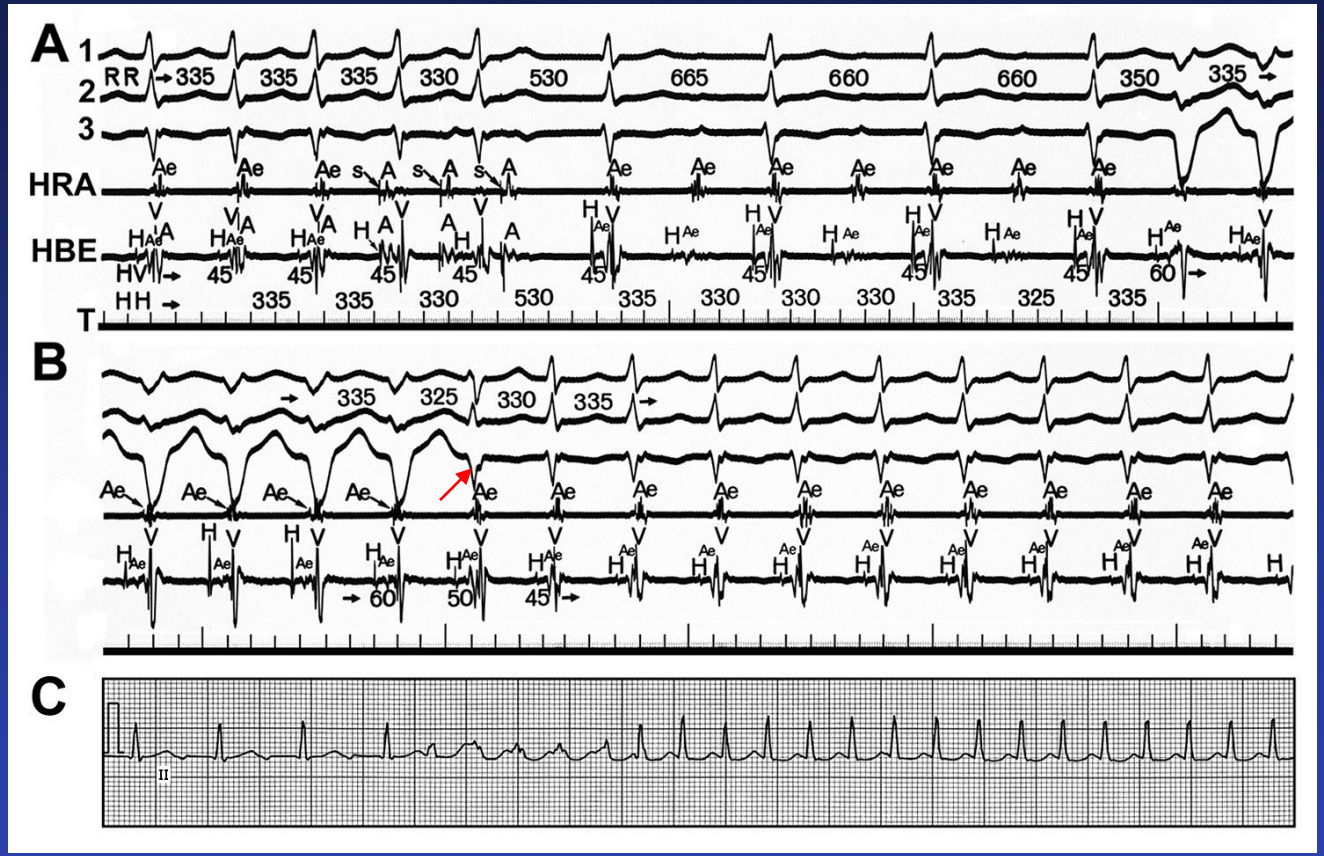
What Cause:

- Changes from slower rate to faster wide complex rhythm
- From faster wide complex to narrow complex rhythm

C

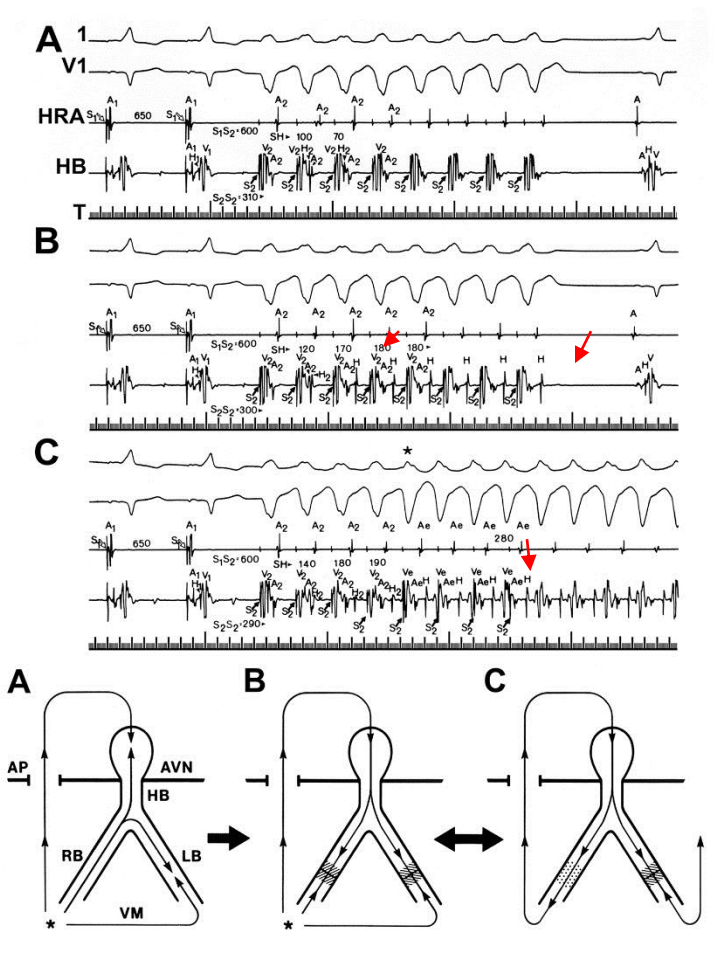


Linking by Inference



- A- 2:1 conduction due to Infra Hisian Block due to short long short sequence in H-H intervals.
- Followed by 1:1 with LBBB pattern due to uneven recovery of BB. Linking is the logical explanation for continuous LBBB
- B.-The site of linking migrates downstream and results in narrow QRS
- Note incomplete LBBB between complete LBBB and narrow QRS

Linking by Interference



Linking by interference and downstream impulse migration in WPW

A1-A1 at 600 ms but S2-S2 changed from 310-290 ms

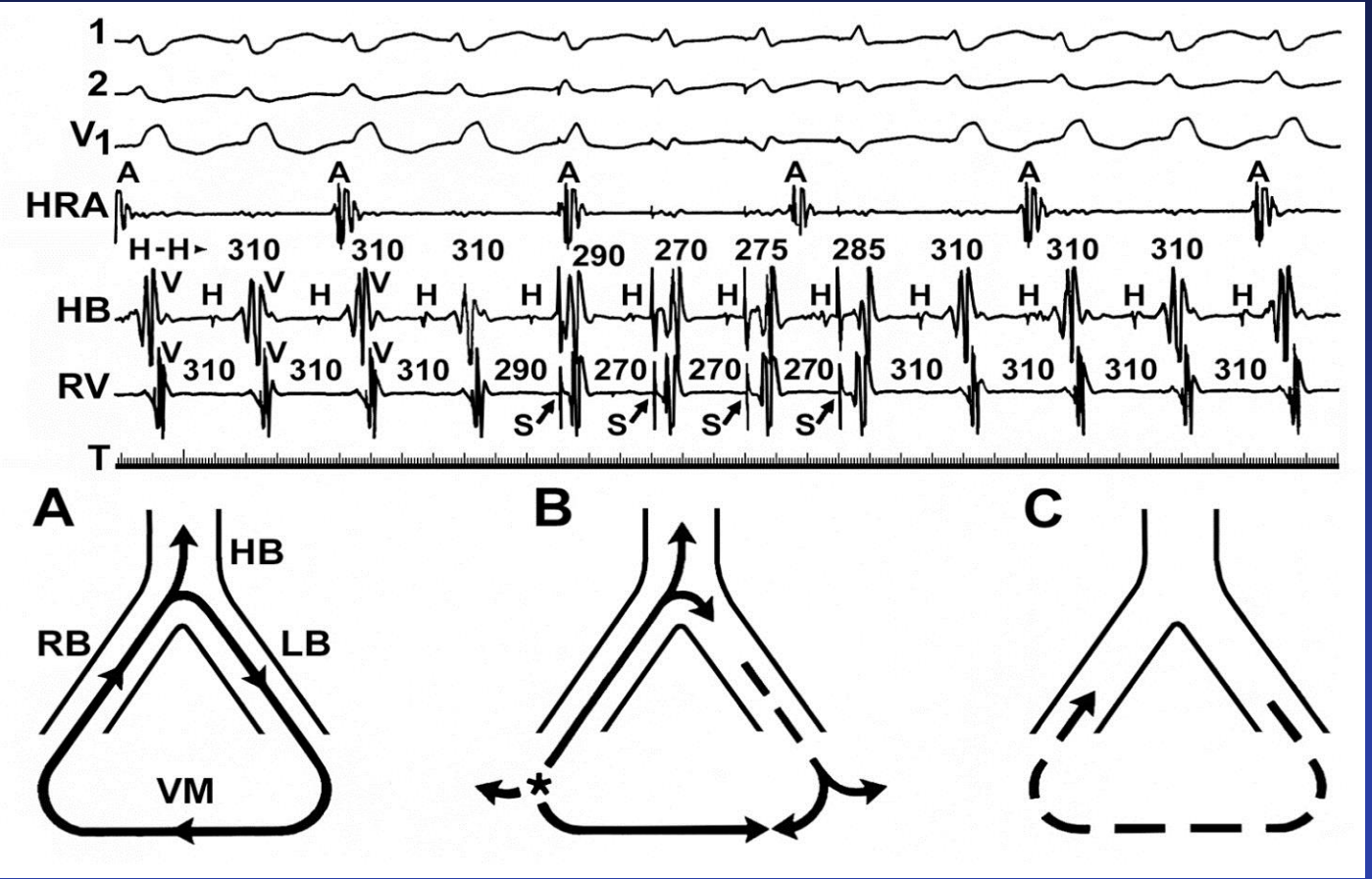
A. -H2 after first S2 obscured by V2
-Second S2 behaves like premature complex where H2 emerges but difficult to identify due to A2 occurring at the same time from AP
-Accommodation than occurs due to rapid conduction over NP and AP

B.- Rapid VH>Slow VH>HPS block which occurs after third S2. His is activated antegrade fashion as A comes early due to conduction over AP and block in HPS

- Linking by interference occurs and continues even after pacing stopped

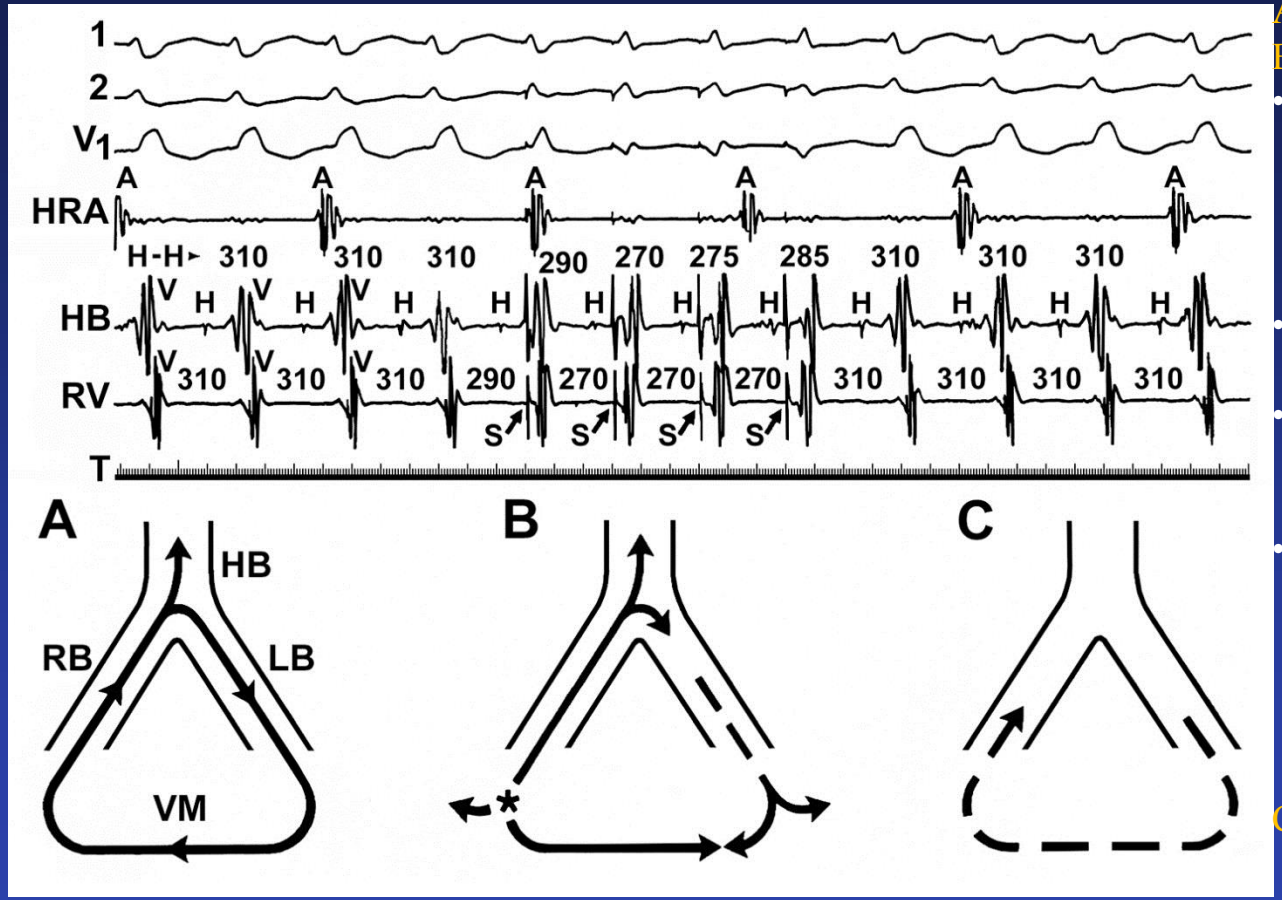
C. -At 290 ms last QRS occurs before stimulus due to forward migration of the block and ORT is initiated with linking in LB

Lehman MH, Akhtar M, Circulation 1985



Lehman MH, Akhtar M, Circulation 1985

Linking by Collision



A-BBR VT with RB pattern

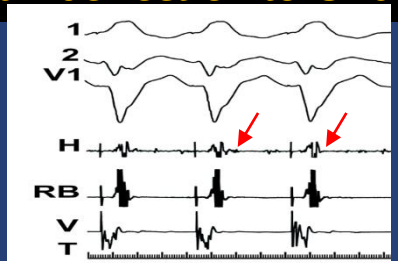
B-Pacing in RV at 270 ms

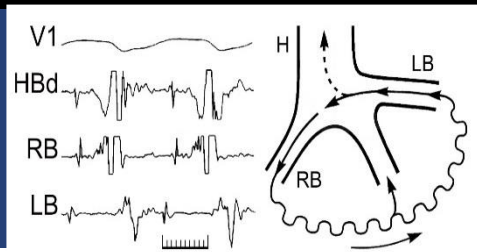
- Travels the same path-retrograde through RB but also travels transeptally colliding with anterograde impulse emerging from LB.
- Paced impulse reaches the His and QRS (resetting)
- QRS activation is via 2 fronts and so is fusion QRS complex (not fully paced or like tachycardia)
- This repeated appearance of QRS (entrainment with fusion) continues with collision of the paced impulse with the tachycardia impulse exiting from LB (linking with collision)

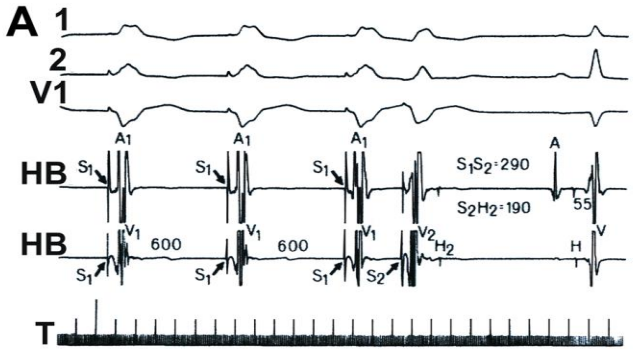
C- Tachycardia resumes with previous CL after pacing is stopped

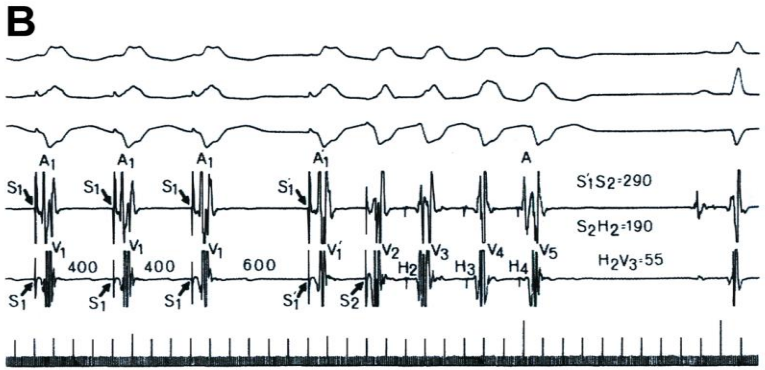
Lehman MH, Akhtar M, Circulation 1985

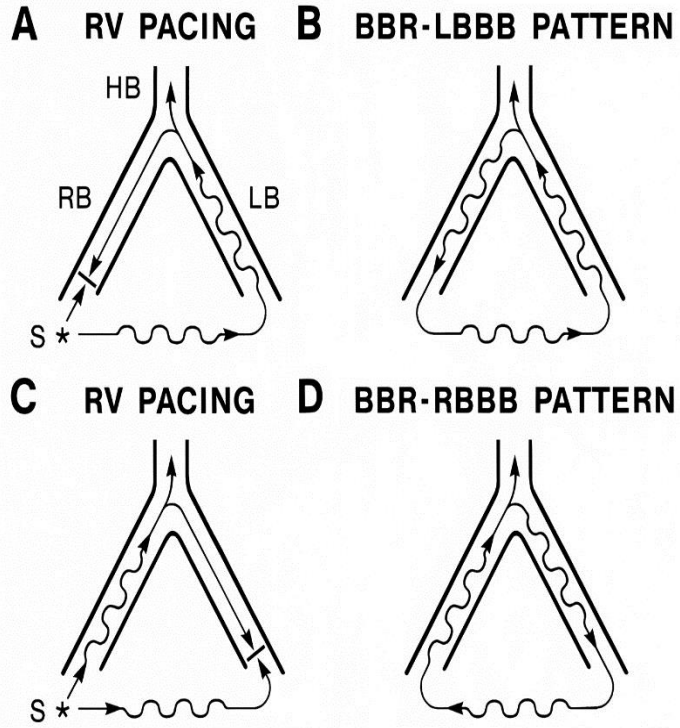
From Small deflection to Understanding His Purkinje Behavior- *A life Spent on Amazing Research*

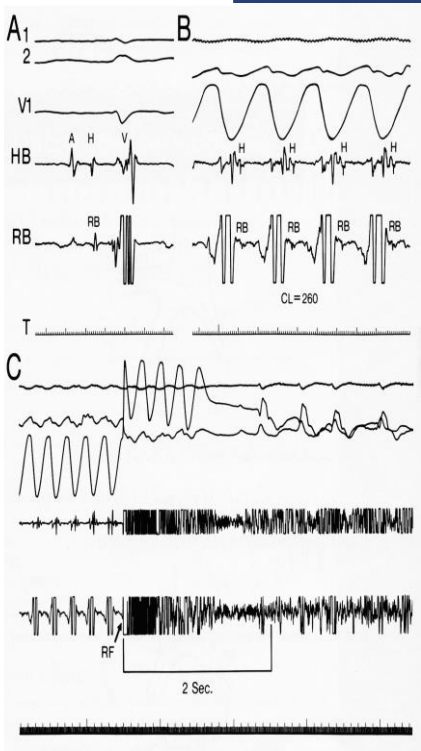












Thank You