WPW:
What's still true?
What's new?

September 2010

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UTHSCSA and STVAHCS

I have no conflicts of interest related to this presentation.

Relevant References

- ACC/AHA/ESC Guidelines for the management of patients with supraventricular arrhythmias, 2003.
- Cardiac Electrophysiology, from Cell to Bedside, 5th ed, 2009. Zipes and Jalife.
- Braunwald's Heart Disease, 8th ed, 2008.

What's Still True?

- How things developed

History of WPW - 1

- 1893: Kent described muscular AV connections, considered them to provide normal AV conduction
- 1914: Mines suggested that Bundle of Kent might mediate reentry
- 1930: Leon Wolff and Paul Dudley White in Boston and Sir John Parkinson in London published 11 cases with bizarre ventricular conduction and short PR intervals "Bundle Branch Block with Short PR Interval in Healthy Young People Prone to Paroxysmal Tachycardia" Am Heart J. 1930;5:685-704.
- 1944: Segers connected short PR interval, wide QRS and prolonged upstroke and arrhythmias into a syndrome

Wolff L. Circulation 1954;10:282; Marriott 9th ed, Ch. 6; Podrid and Kowey, Ch 17, 2001.

RESEARCHES ON THE STRUCTURE AND FUNCTION OF THE MAMMALIAN HEART. By A. F. STANLEY KENT, M.A., *Magdalen College, Oxford. Assistant to the Waynflete Professor of Physiology in the University of Oxford.* (Pl. XII.)

- "Briefly the phenomena in question are concerned with the passage of the wave of contraction over the auriculo-ventricular groove, an explanation being required of the mode in which an auricular contraction is able on arriving at the groove to initiate a contraction of the ventricle"

Journal of Physiology, March 1893.

Dr. Kent's Data

- AV groove in monkey. "At the lower part of the figure on the right, a stellate mass of auricular muscle is seen, some of the fibers of which become continuous with some of the scattered branched muscle cells lying in the fibrous tissue."

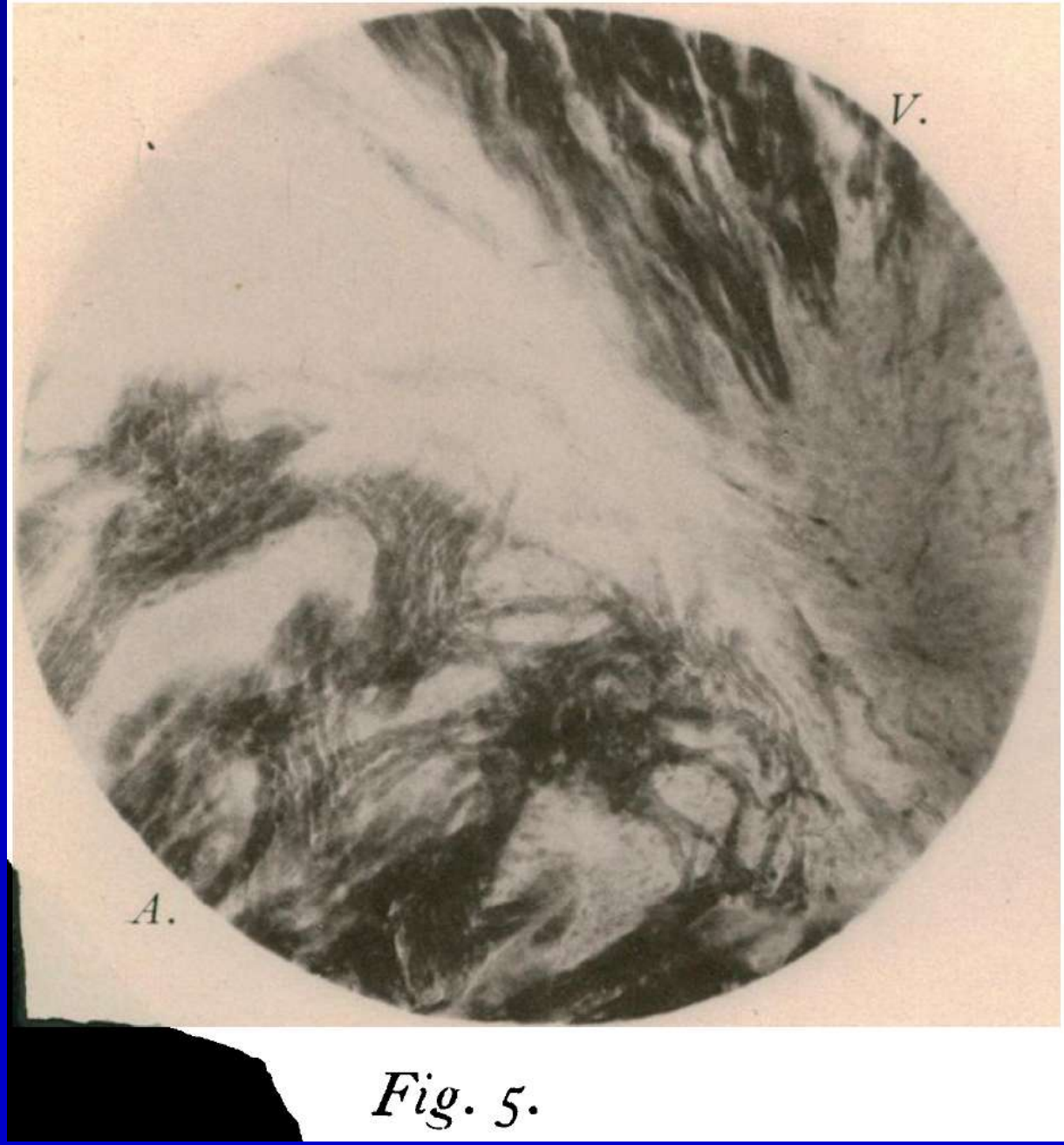


Fig. 5.

Journal of Physiology, March 1893.

Dr. Kent's Conclusion

- "The passage of the contraction over the cardiac tissue of the heart then appears to occur as a simple muscular wave, and the transmission of the contraction across the auriculo-ventricular groove appears to be of a similar nature."

Journal of Physiology, March 1893.

Original Article

The American Heart Journal

VOL. V AUGUST, 1930 No. 6

Original Communications

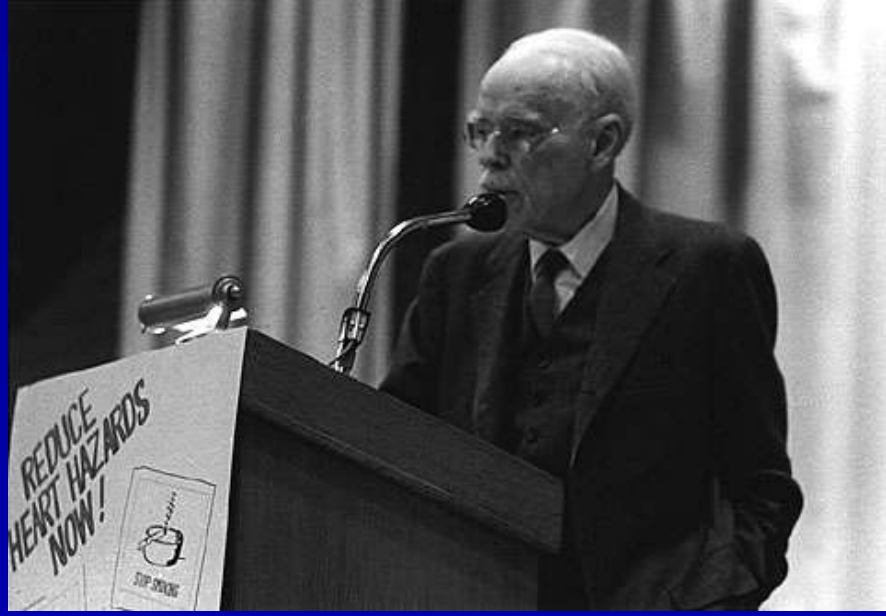
BUNDLE-BRANCH BLOCK WITH SHORT P-R INTERVAL IN HEALTHY YOUNG PEOPLE PRONE TO PAROXYSMAL TACHYCARDIA

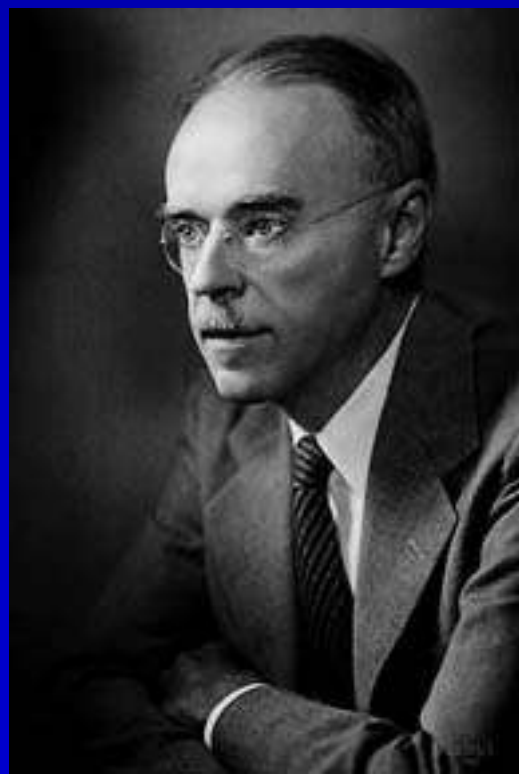
LOUIS WOLFF, M.D., BOSTON, MASS., JOHN PARKINSON, M.D., LONDON, ENG., AND PAUL D. WHITE, M.D., BOSTON, MASS.

The Three Original Authors

Paul Dudley White

1886-1973





WPW Exercise, Atropine

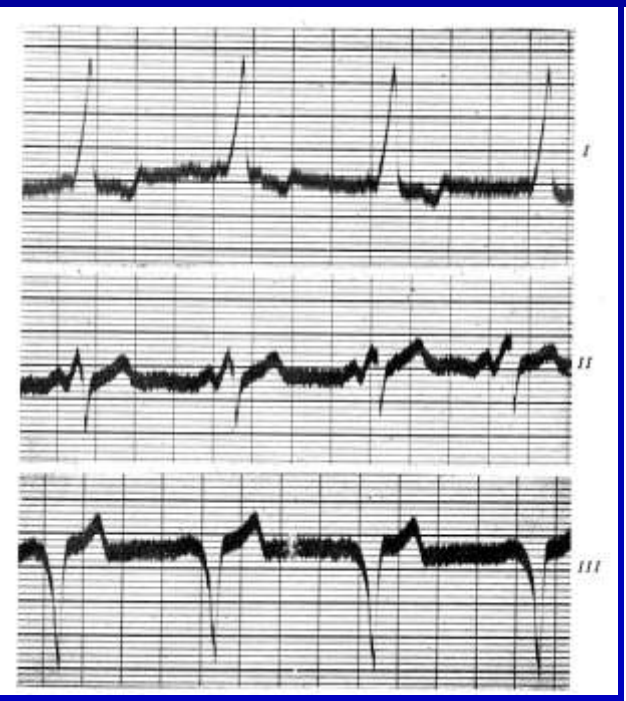


Fig. 1.—(Case I) Right bundle-branch block. The P-R interval is 0.1 second. The rate is 72. Time intervals for this and succeeding figures = 0.2 second. Horizontal lines cut off intervals of 10^{-4} volt.

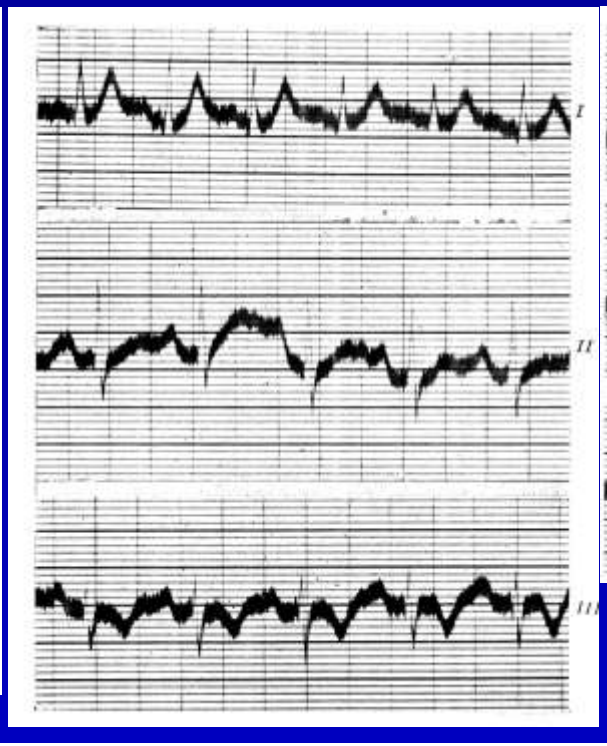


Fig. 2.—(Case I) Immediately after exercise (running up and down four flights of stairs). Sino-auricular tachycardia, rate 140 to 120. The ventricular complexes are normal, the P-waves are better marked, and the P-R interval is 0.16 second.

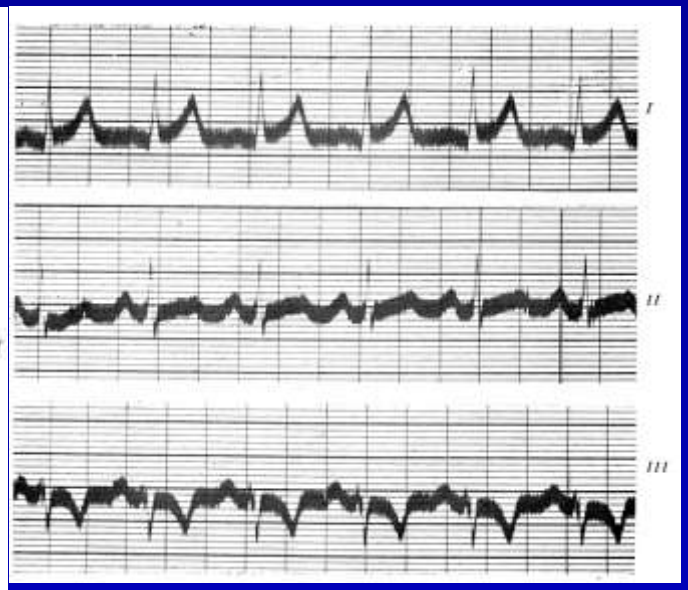


Fig. 3.—(Case I) One hour after the subcutaneous injection of 1/30 grain of atropine sulphate. The rate is 140, the ventricular complexes are normal, and the P-R interval is 0.15 to 0.16 second.

WPW and Exercise

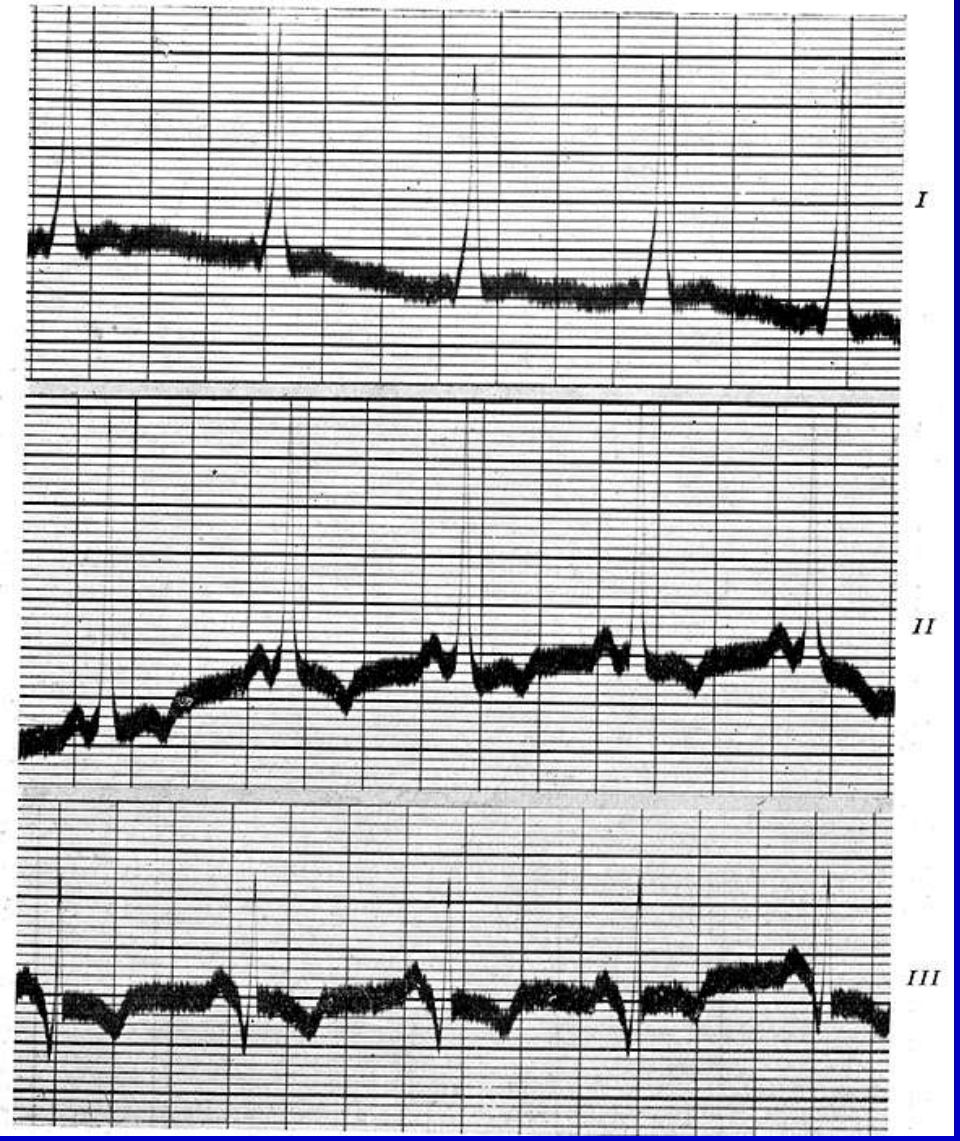


Fig. 4.—(Case II) Intraventricular block. The P-R interval is 0.1 second. Rate 96.

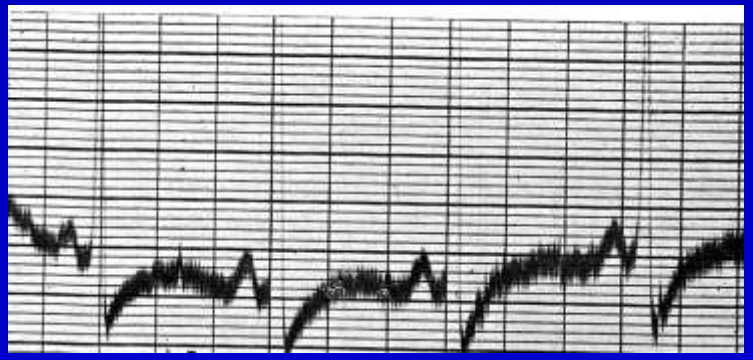


Fig. 5.—(Case II) Immediately after exercise. The ventricular complexes are normal except for deformity of the S wave and S-T interval by artefact (high resistance, resulting in over-shooting). The T-wave is upright. The P-R interval is 0.15 second Rate 96.

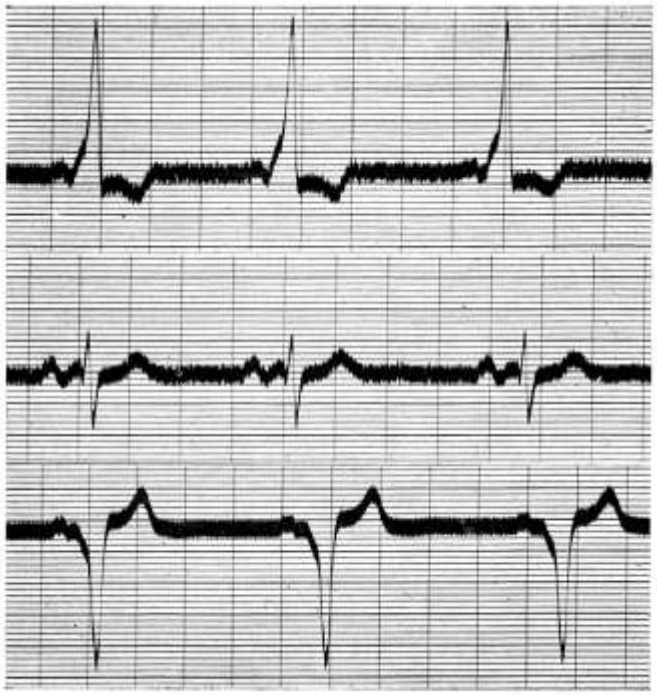


Fig. 6.—(Case III) Right bundle-branch block. The P-R interval is well under 0.1 second. The rate varies between 60 and 70.

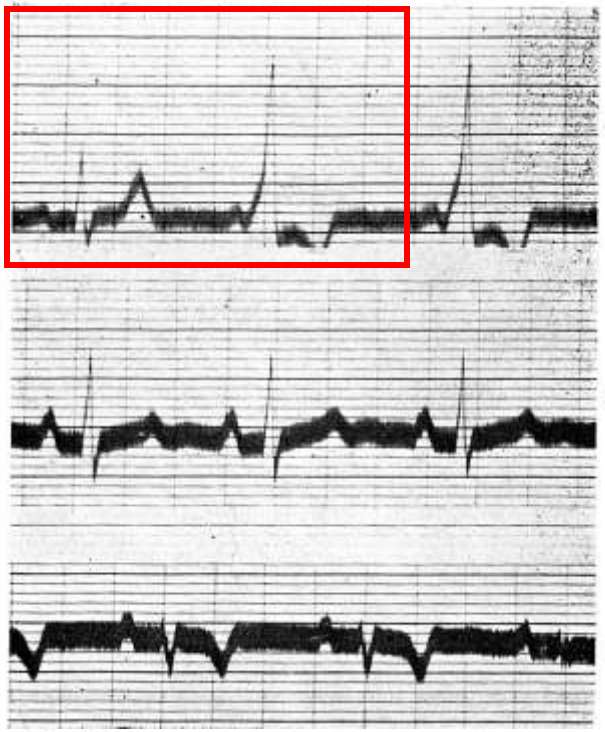


Fig. 7.—(Case III) After a paroxysm of tachycardia lasting seven hours. The ventricular complexes are normal, but occasionally there is reversion to the abnormal form. The P-R interval is almost 0.2 second. The P-waves are notched, and identical in Figs. 6 and 7.

Fig. 8.—(Case III) Two years after Fig. 6 was taken. Normal physiological curves.

Original Article, Case III, Intermittent Pre-excitation

Original Article, Case IV, Intermittent

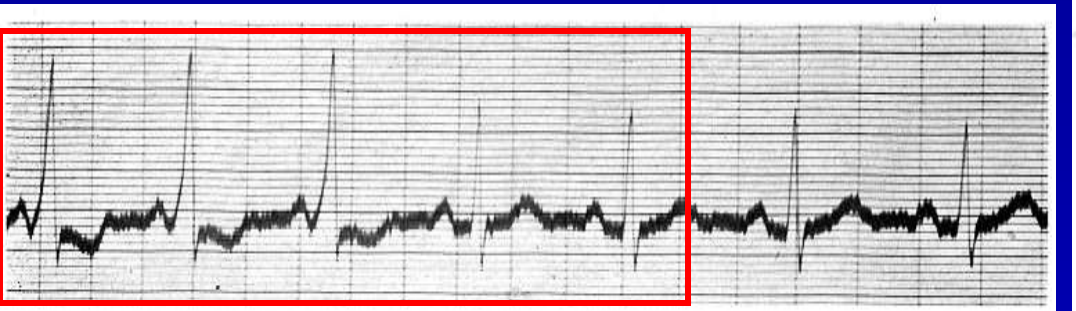


Fig. 9.—(Case IV) Spontaneous reversion from bundle-branch block curves to normal ones. The form of the P-wave remains unaltered, but the P-R interval changes from 0.09 second to 0.15 second.

Fig. 10.—(Case IV) Normal physiological complexes. P-R interval 0.15 second. In Lead II there is a transition to the abnormal form and short P-R interval.

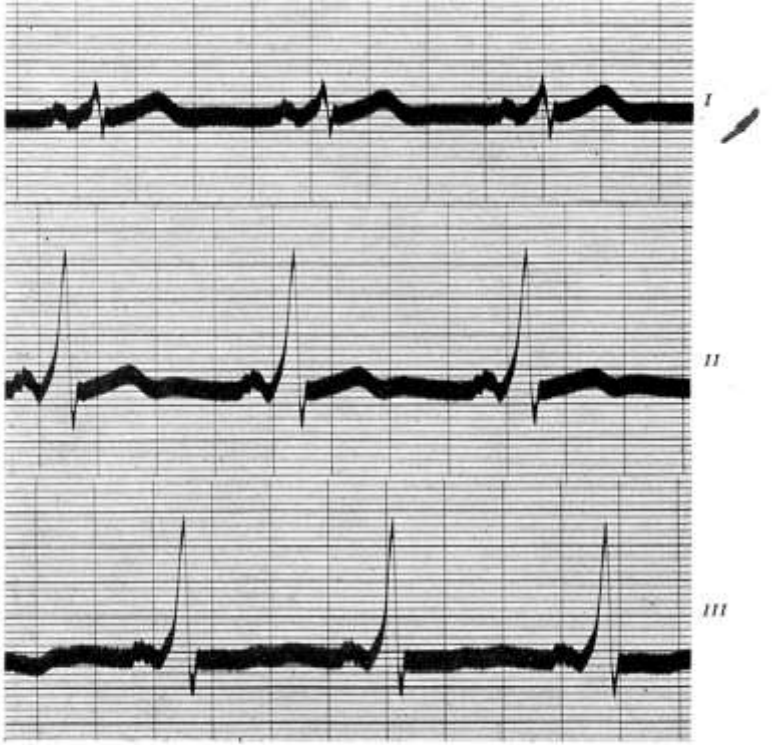


Fig. 11.—(Case IV) Bundle-branch block. The P-R interval is less than 0.1 second. The P-waves are identical in Figs. 10 and 11; note the peculiar notching of the P-waves.

Fig. 12.—(Case IV) Simultaneous electrocardiogram and jugular and radial tracings. Bundle-branch block curves are present. The a. c. v. h. sequence is normal.

History of WPW - 2

- 1940 Richard F Ohnell (Cardiologia 1940) described a patient with WPW and SCD

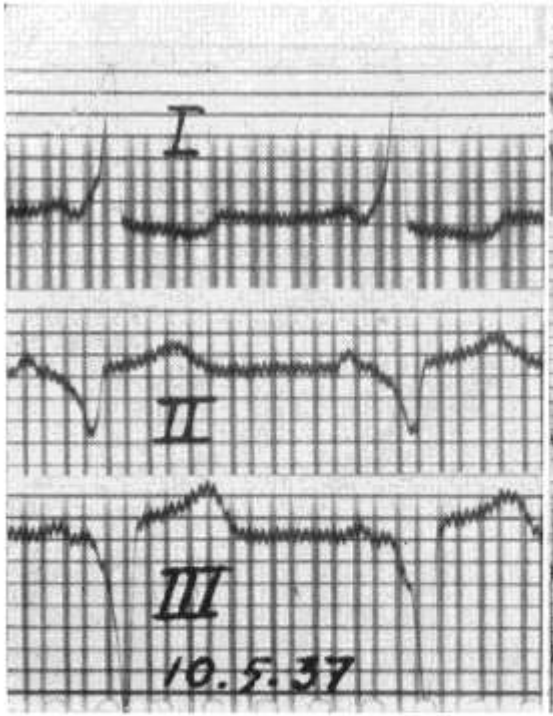


Fig. 1. Timemark. 0.10″ between every second line. PQ in lead II: 0.08″, QRS in lead II: 0.13″.

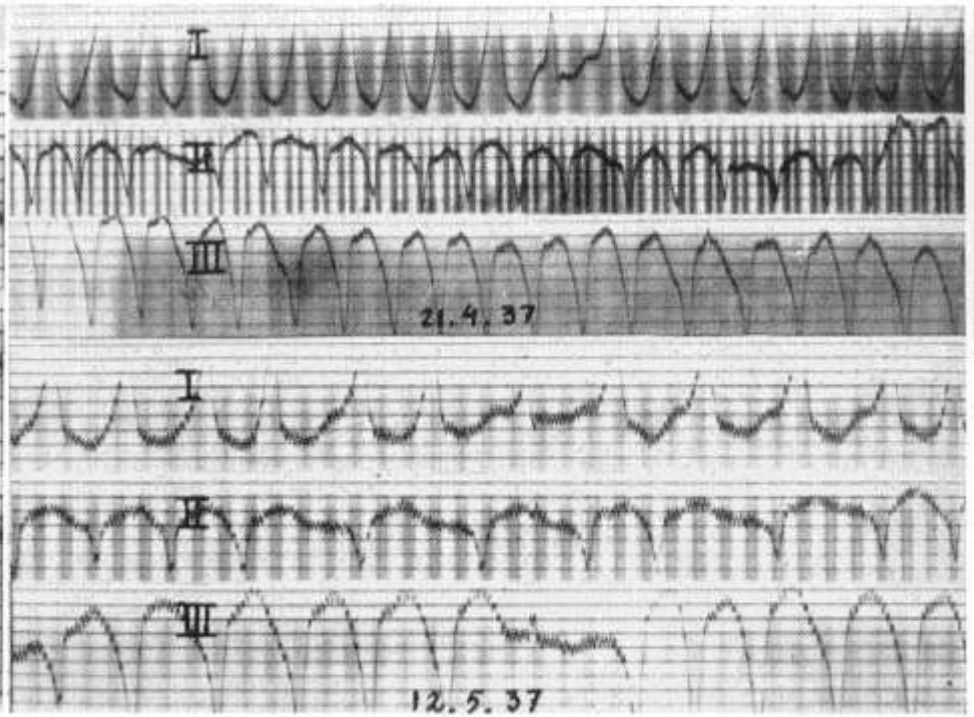
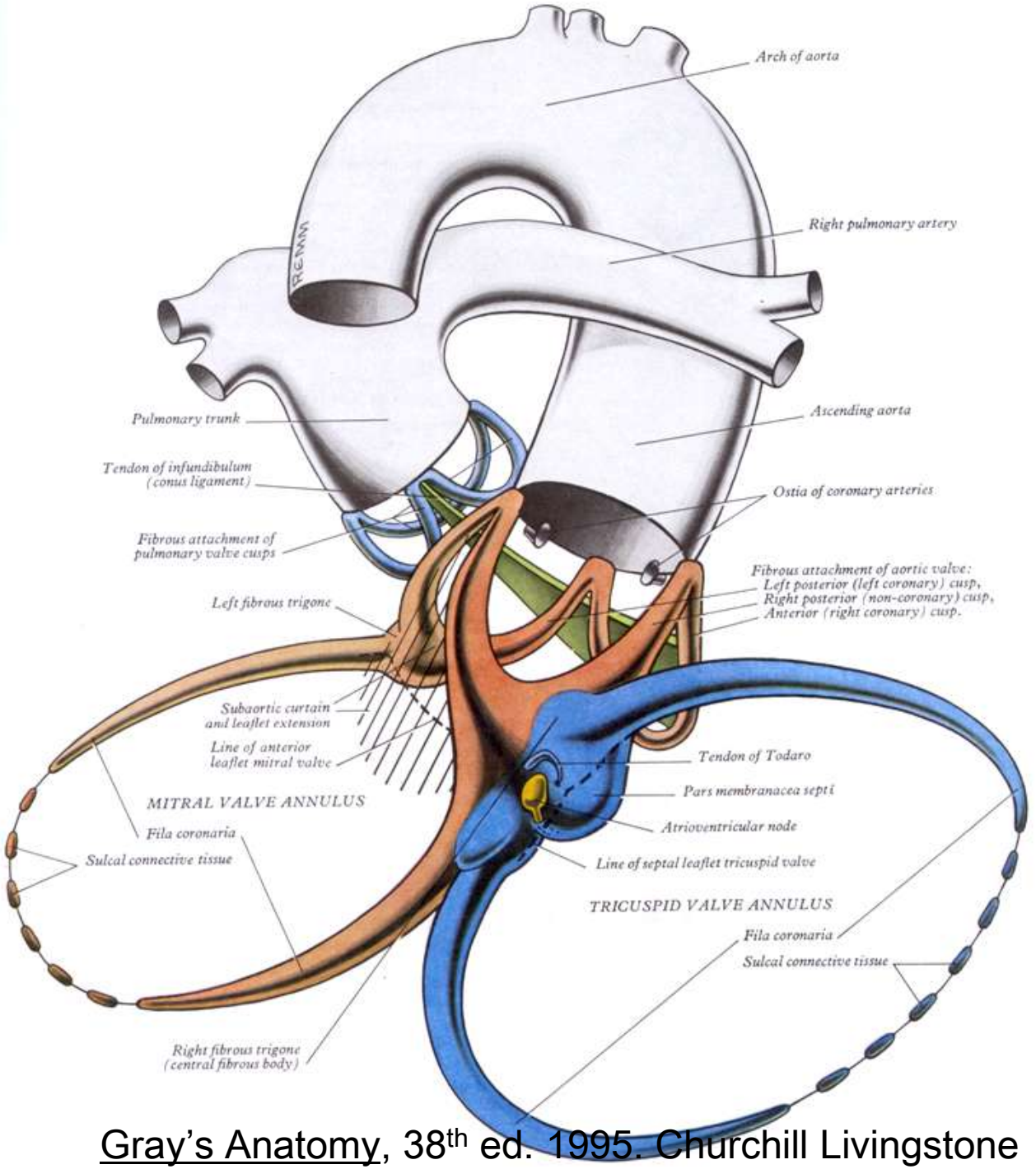


Fig. 2. Timemark. 0.10″ between beginning of every second dark line. Ecg. from two attacks.—Above: Frequency sometimes 285/min.—Below: Rhythm more irregular: Paroxysmal fibrillation—ventricular fibrillation—flutter? Rate aver. 170/min.

Fibrous Skeleton of the Heart

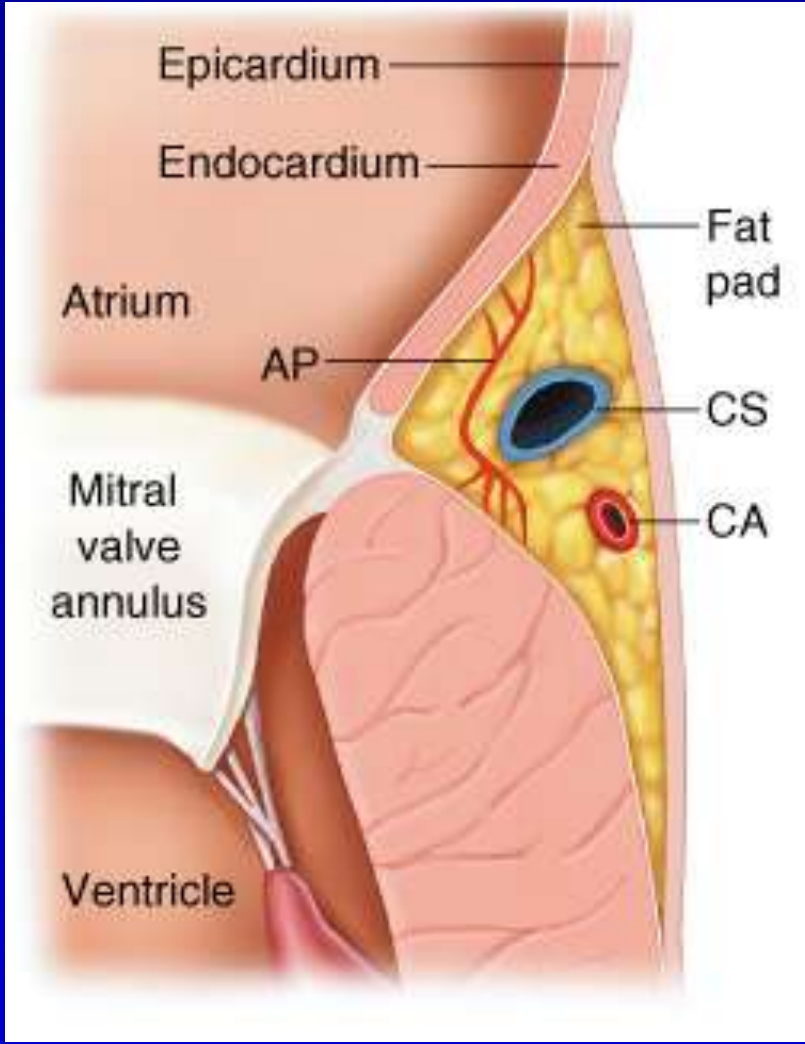
Posterior view
The mitral annulus is generally thicker, more robust and fibrous than the tricuspid annulus



Gray's Anatomy, 38th ed. 1995. Churchill Livingstone

Ventricular Pre-excitation

- 1/500 individuals
- Residual tissue after segmentation of the embryonic cardiac tube into atrial and ventricular chambers
- Fibers usually resemble ordinary myocardium*
- The fiber course from atrium to ventricle may be oblique to the long axis of the ventricle



Zipes and Jalife, 5th ed, 2009, p. 605-613; Braunwald, 2008, p. 819. *Peters NS et al. Eur Heart J. 1994;15:981.

WPW and Structural Disease

- Most have structurally normal hearts
- Some have abnormalities
  - Ebstein's anomaly
  - HCM
  - MV prolapse
  - Genetic abnormalities PRKAG2 mutation (cardiac structure)
  - Mutation in BMP gene (neurologic)

Pathway Locations

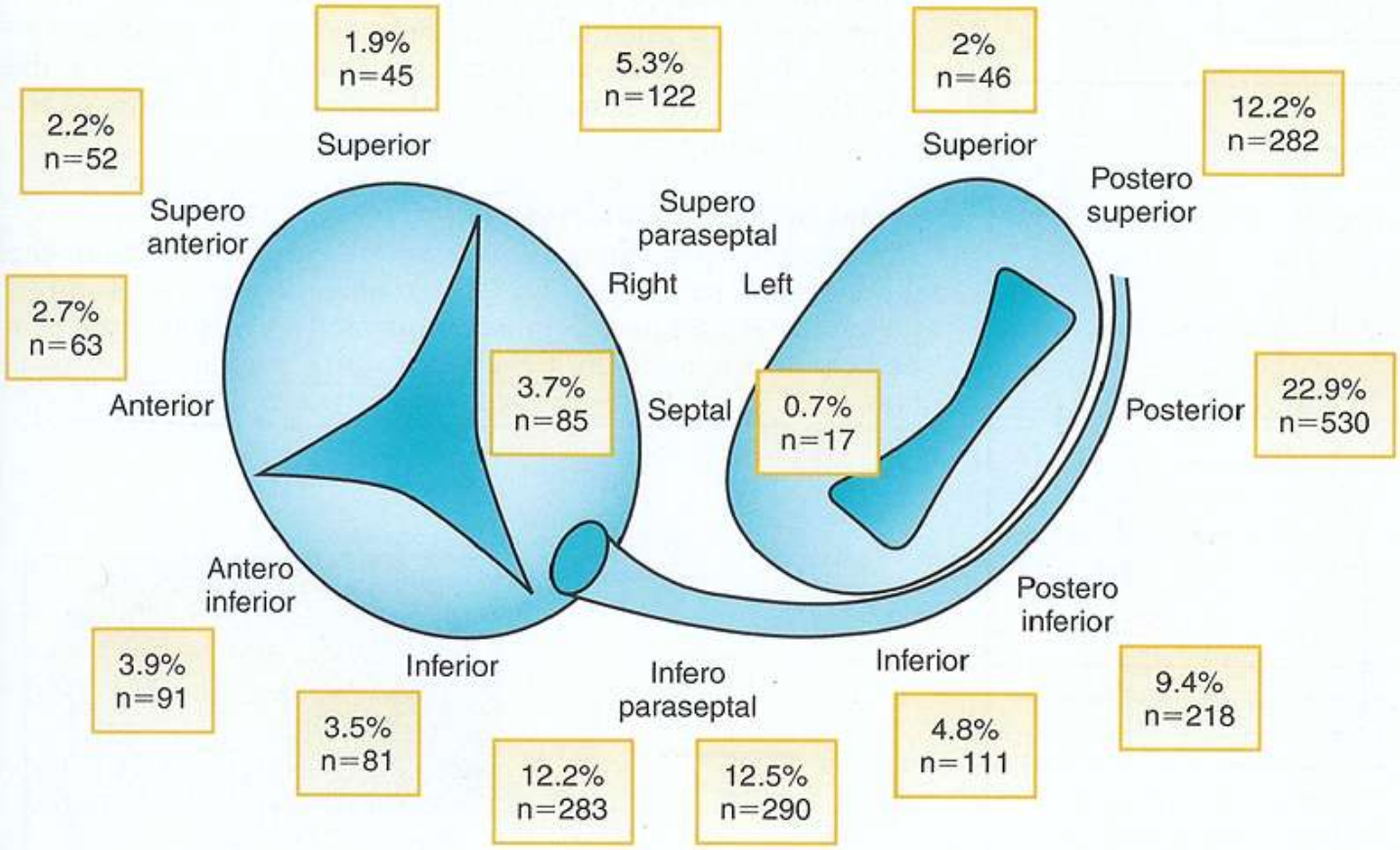
n=1969 patients (1146 m)
Mean age 36±18 years

	Patients	Ebstein
	1969 2325 APs	40/1969 86 APs
1 AP	1705	12
2 APs	193	15
3 APs	57	10
4 APs	8	1
5 APs	5	2
6 APs	1	0

60% left free wall

25% septal

15% right free wall



Zipes, 5th ed. P. 1085

ECG Diagnosis

- Short PR interval
- Delta wave (slurred upstroke)
- Long QRS
- ST-T abnormality generally discordant from the delta wave and main QRS
- More subtle in left free wall pathways (the most common)
- Tachyarrhythmia is required for WPW syndrome

ECG Localization of Pathway

- There are many classification schemes by ECG
- All require manifest pre-excitation and up to 50% of pathways conduct only retrograde (“concealed”)
- None is perfect
- I leave the details to the electrophysiologist

Localizing the Accessory Pathway - 1
(To be sure, must do EP study)

- Left free wall: negative delta wave in I, aVL, or V6 and "pseudo-RBBB" with Rs in V1
- Right anteroseptal (early ventricular activation near His bundle): positive delta wave in 2, 3, aVF, and low R/S in V1-V3 and late R wave transition
- Posteroseptal: negative or isoelectric delta waves in 2, 3, aVF and rapid R wave transition V1-V2

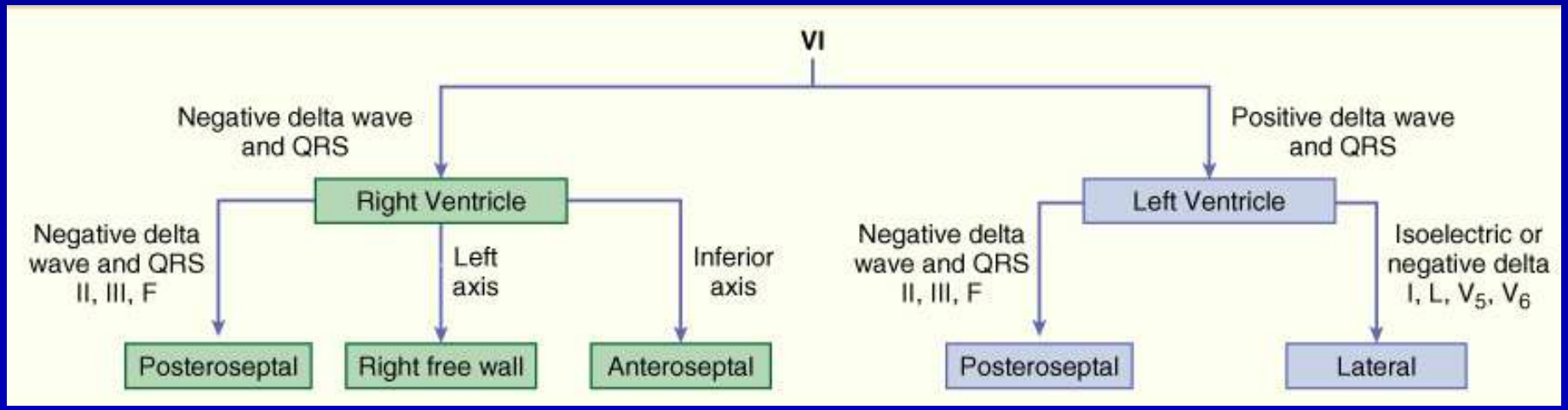
Zipes and Jalife, 4th ed, 2004, p. 869-878

Localizing the Accessory Pathway - 2
(To be sure, must do EP study)

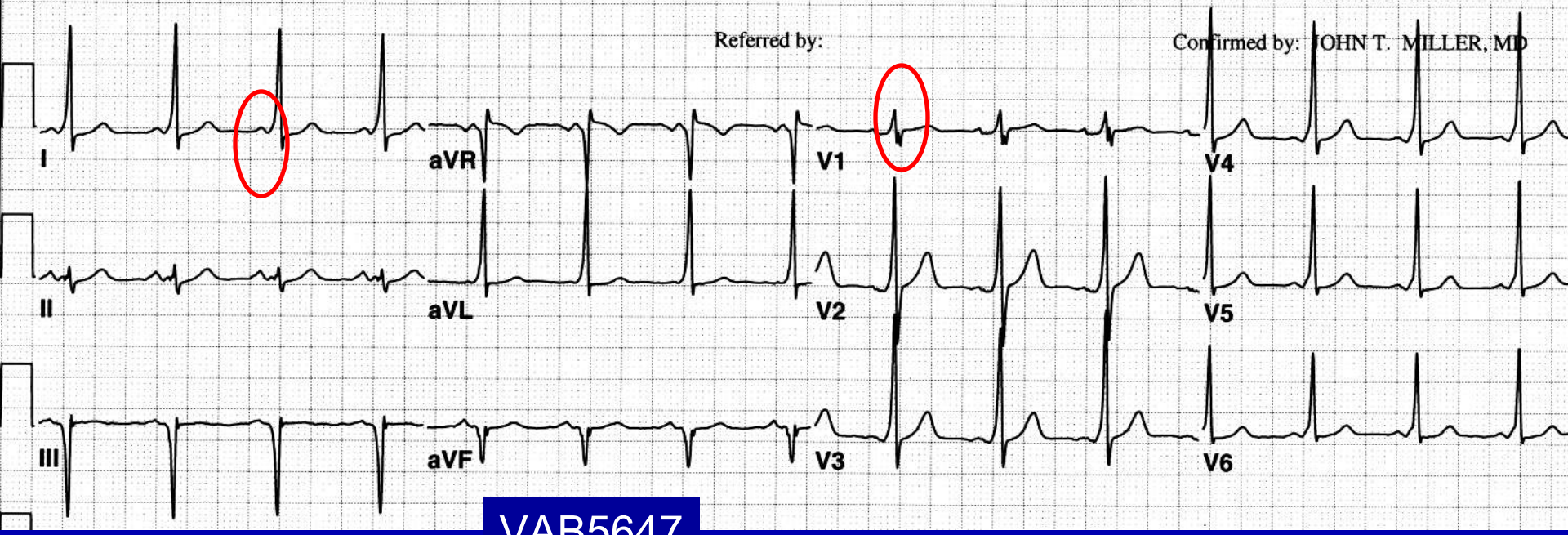
- Right free wall: positive delta wave in I and pseudo-LBBB
- Generally loss of a positive delta wave from leads 3 to aVF to 2 as the pathway location moves from anterior septal to posterior septal site around either AV ring
- For right-sided pathways, a positive delta wave occurs sequentially in V1 to V4 as the pathway location moves from anterior to posterior around the TV ring

Zipes and Jalife, 4th ed, 2004, p. 869-878

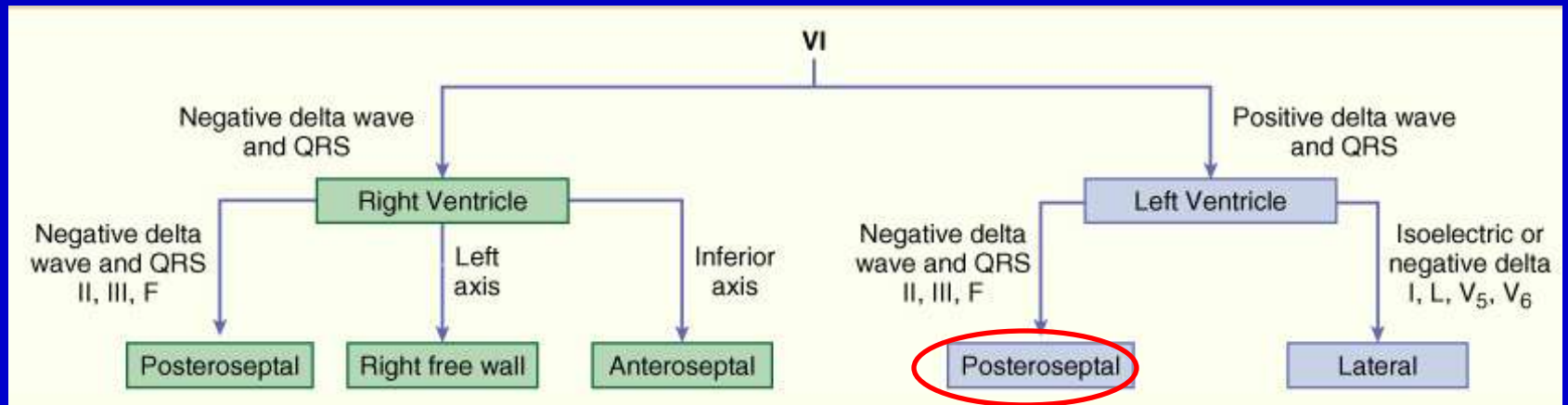
Localization of Bypass Tract



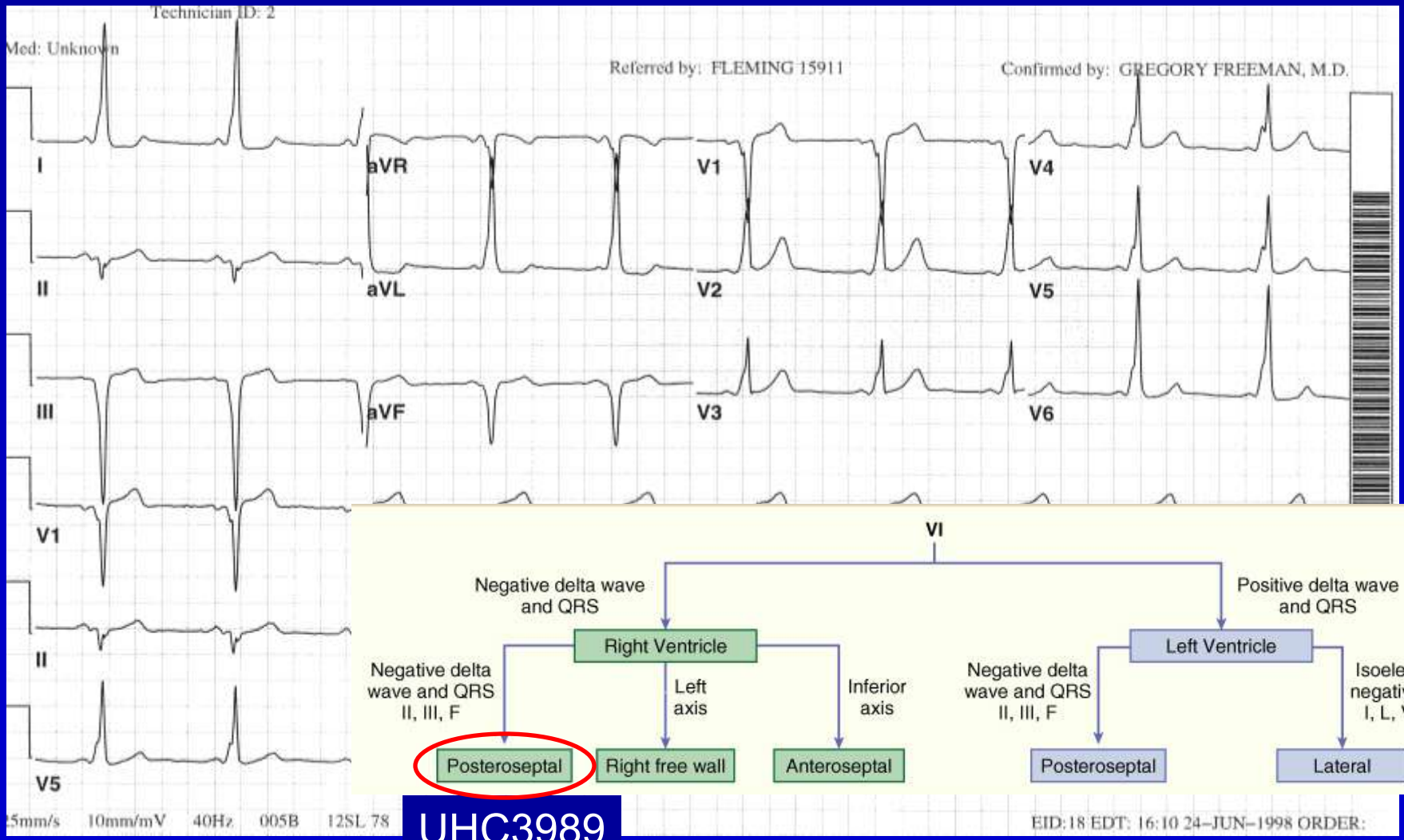
Braunwald, Ch. 32, “Specific Arrhythmias: Diagnosis and Treatment”, Olgin JE and Zipes DP. p. 830, 2005



Left posterior or paraseptal



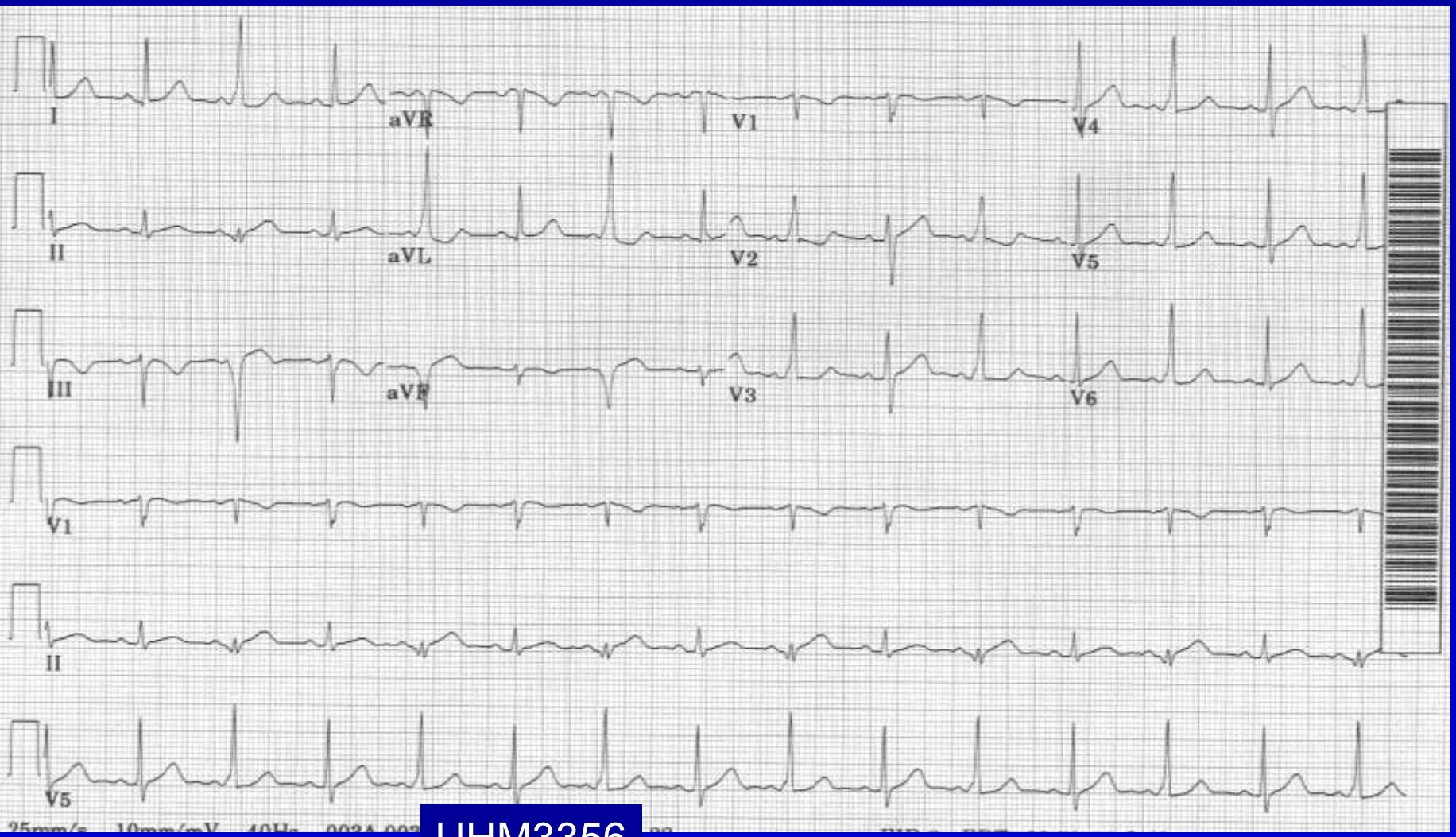
Right posteroseptal – sudden transition from V1-V2, negative in II, III, and aVF



UHC3989

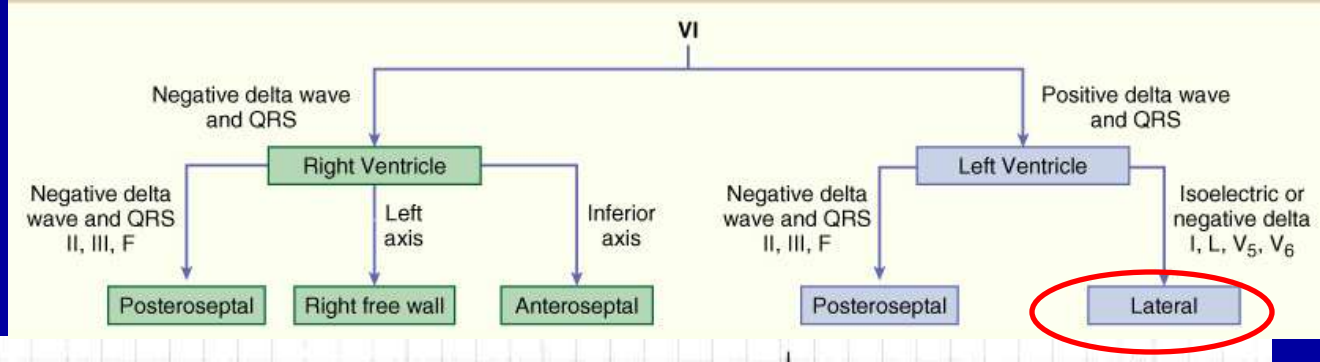
Intermittent preexcitation implies long refractory period of AP, so low risk of AF-RVR-VF

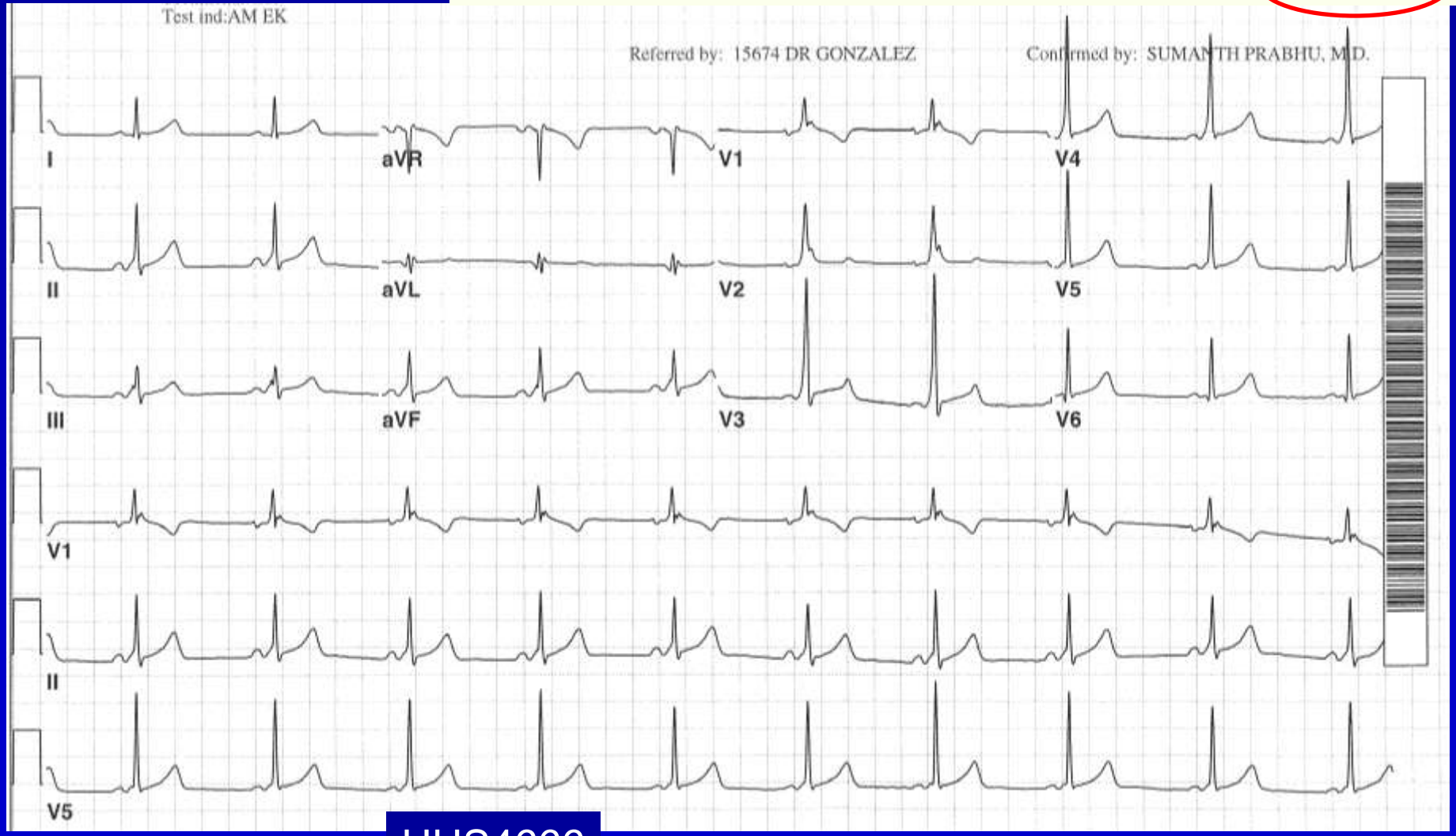
Right posterior paraseptal



UHM3356

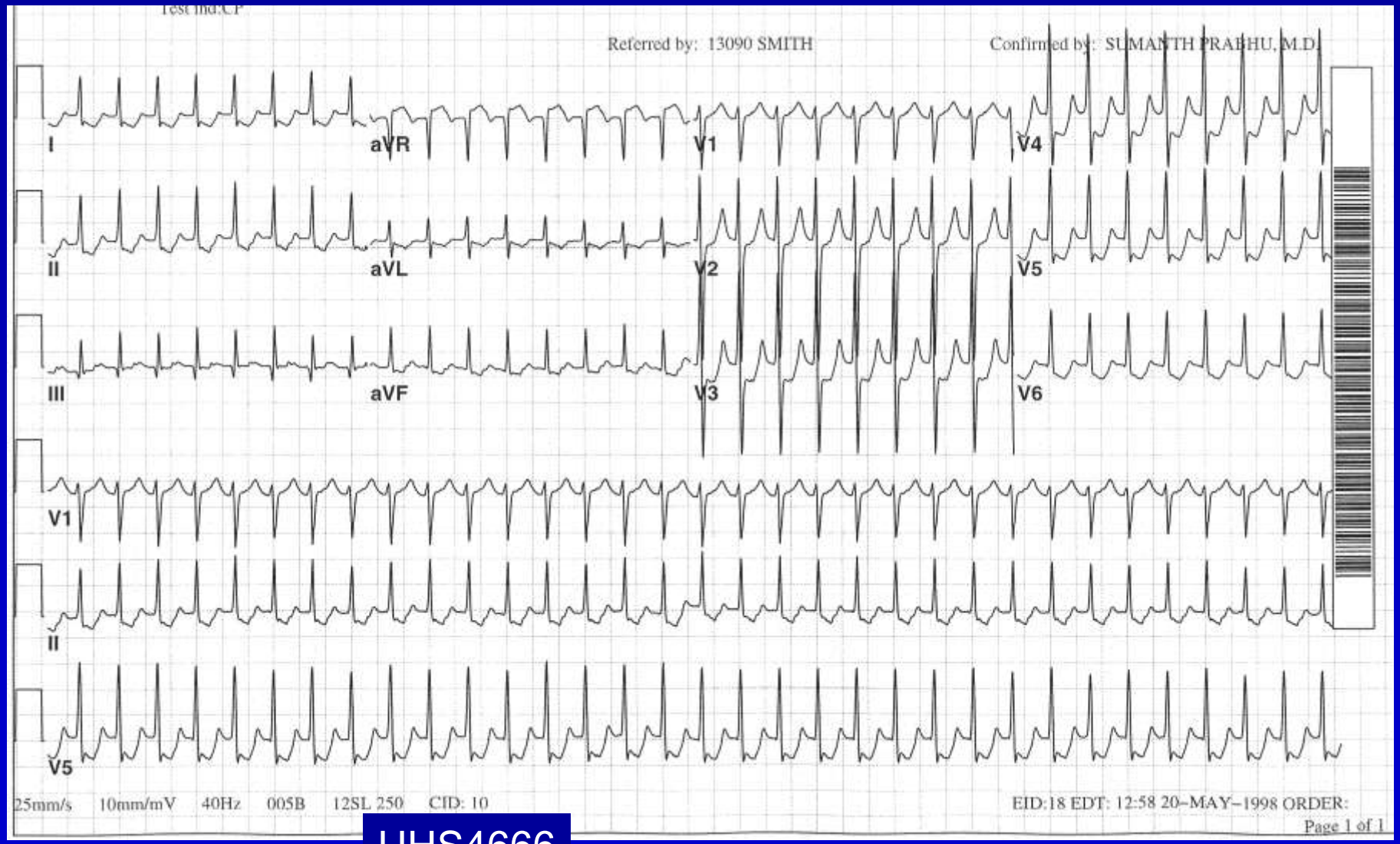
Left lateral





UHS4666

Left lateral, patient with orthodromic AVRT
No preexcitation, P following QRS onset by 0.13 sec,
P upright in inferior leads



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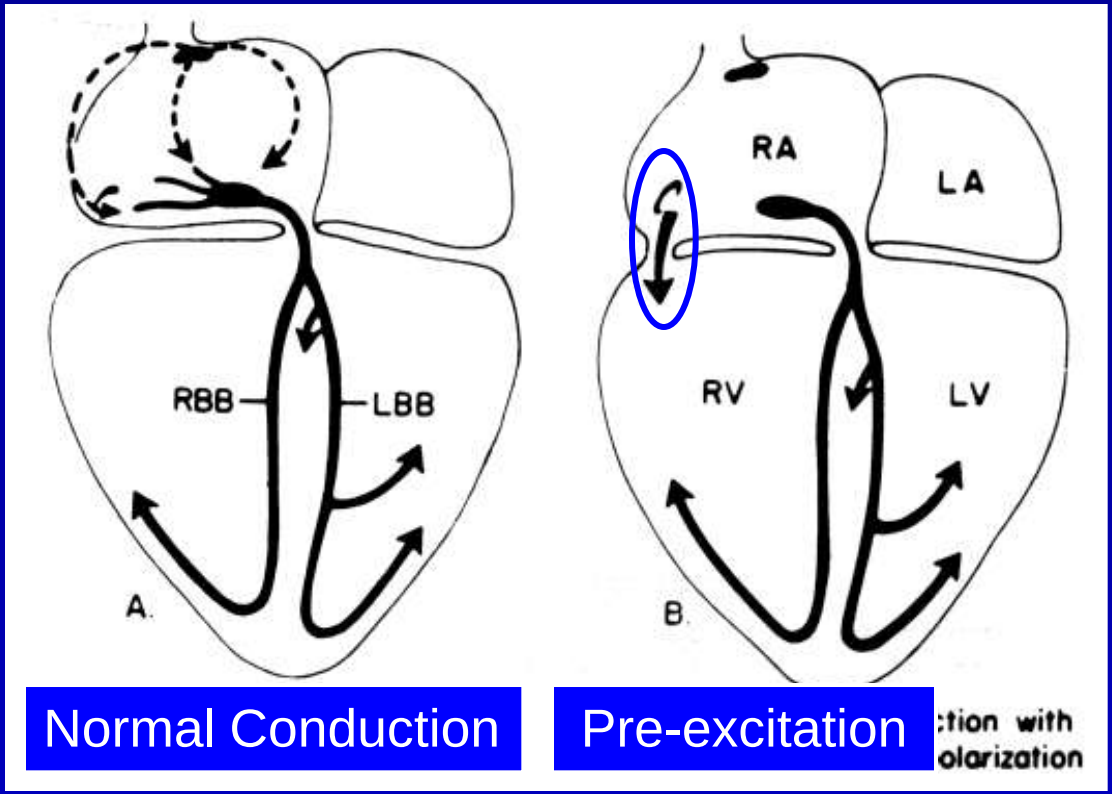


WPW

From Surawicz; Chou, 2001, p. 472

Orthodromic AVRT

- Most common tachycardia in WPW

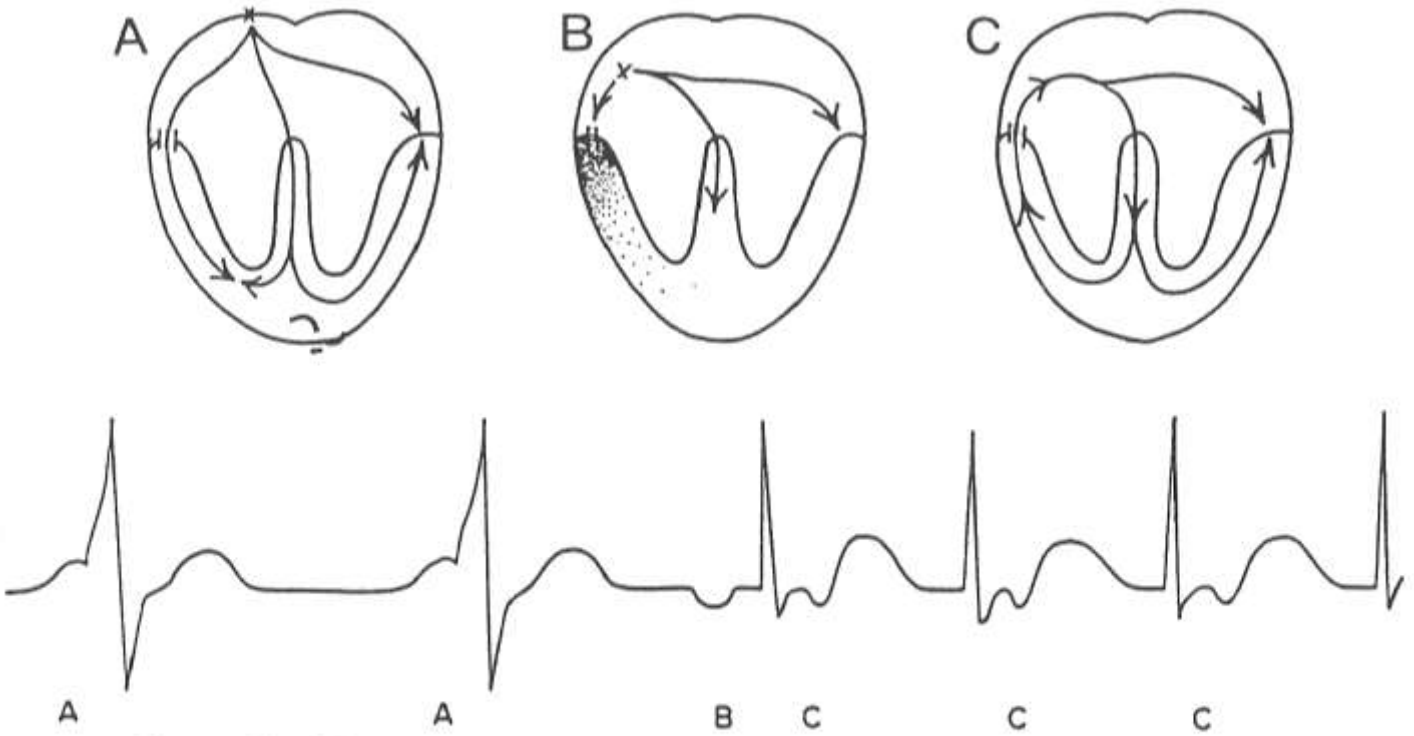


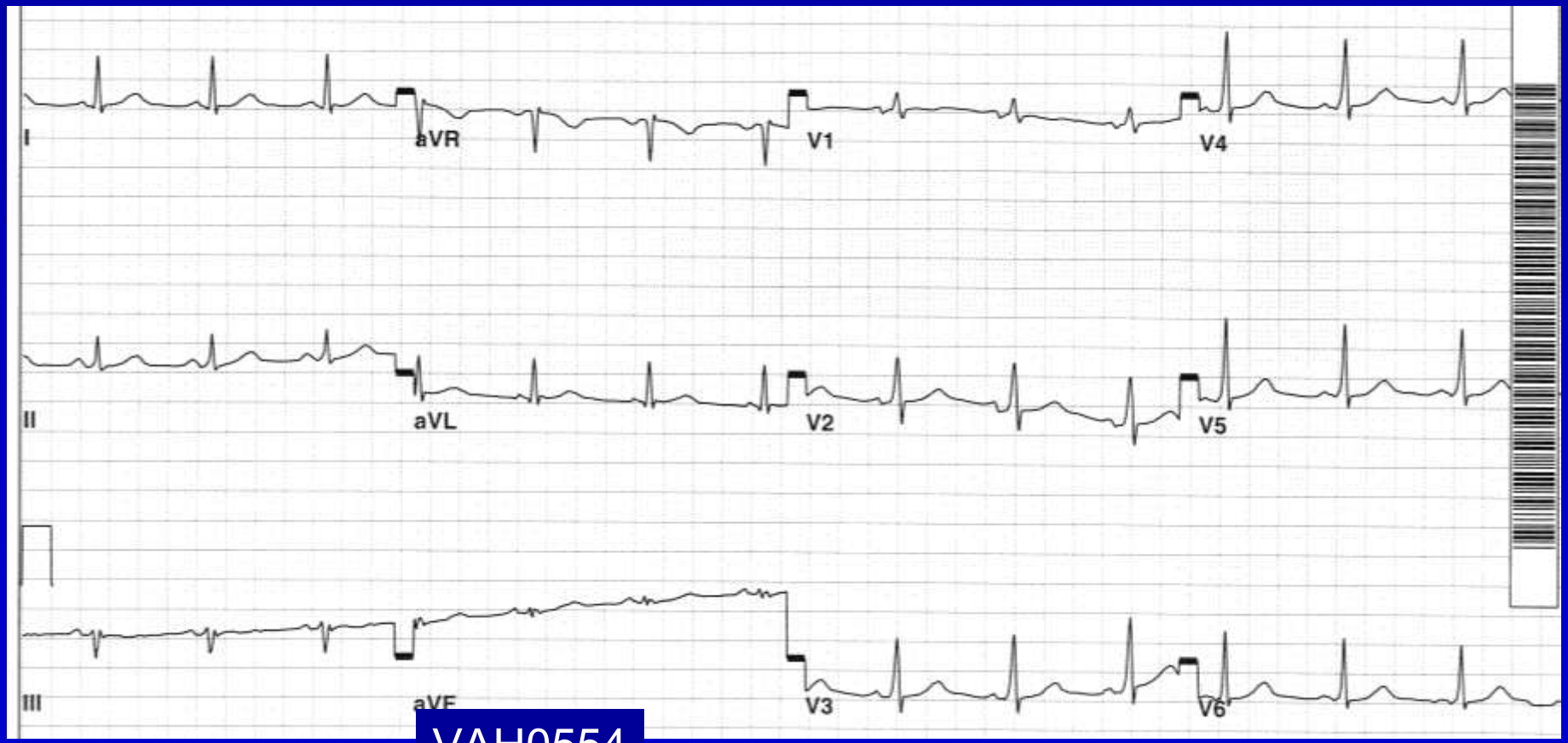
Figure 6.6. The schematic illustration from Figure 6.3 is repeated on the *left* (**A**). In **B**, an atrial premature beat occurs before the Kent bundle has completed its period of refractoriness following the previous sinus beat, preventing antegrade ventricular preexcitation. Normal ventricular activation is then followed by retrograde atrial excitation (**C**). The resultant macroreentrant circuit forms the basis for the tachyarrhythmia. (From Wagner GS, Waugh RA, Ramo BW. Cardiac arrhythmias. New York: Churchill Livingstone, 1983:13.)

Marriott, 9th ed, p. 107

Orthodromic AVRT

- If P wave is visible
  - Inverted in I – left lateral accessory pathway
  - Unfortunately, frequently impossible to discern

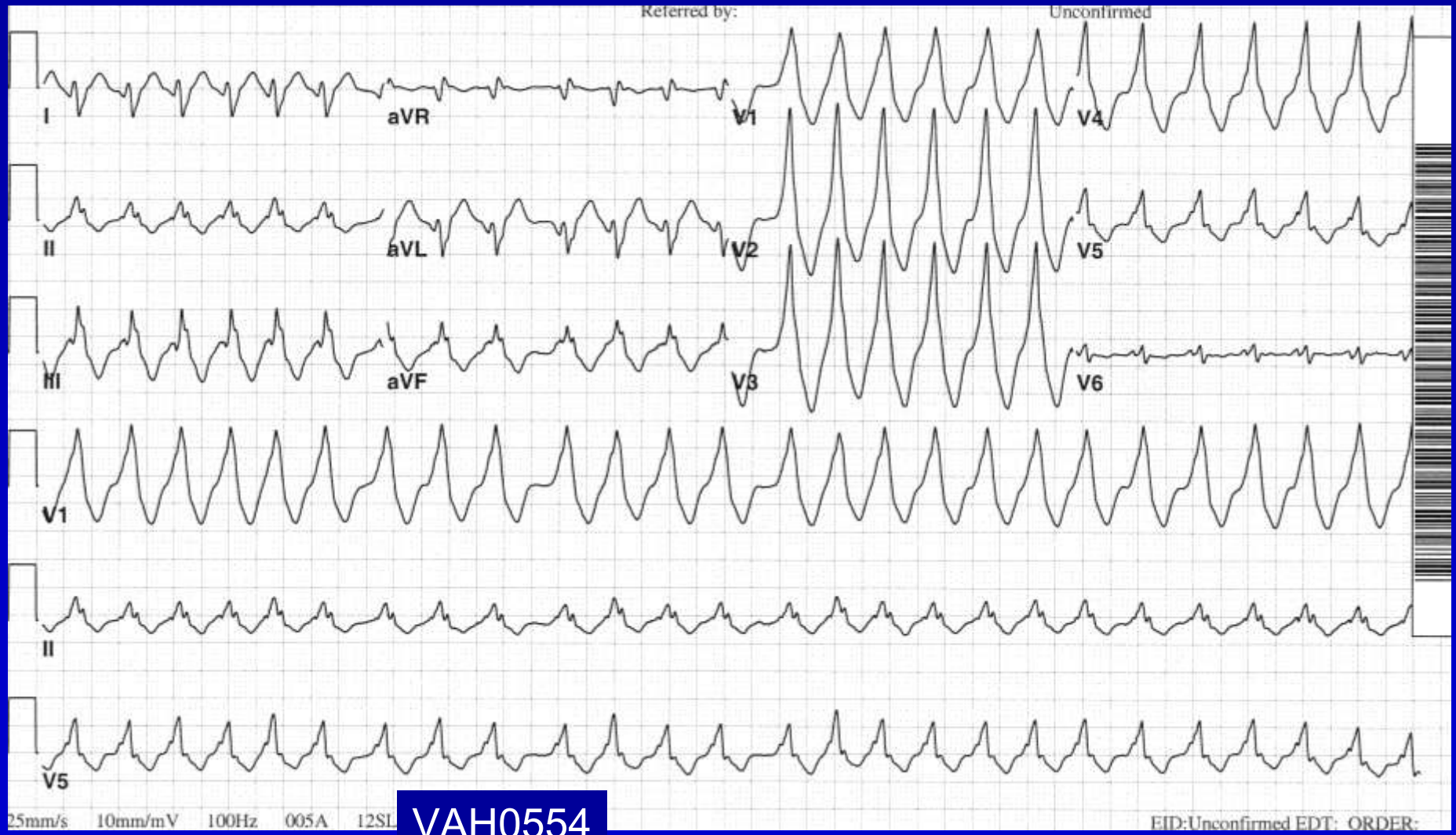
Left lateral or anterolateral



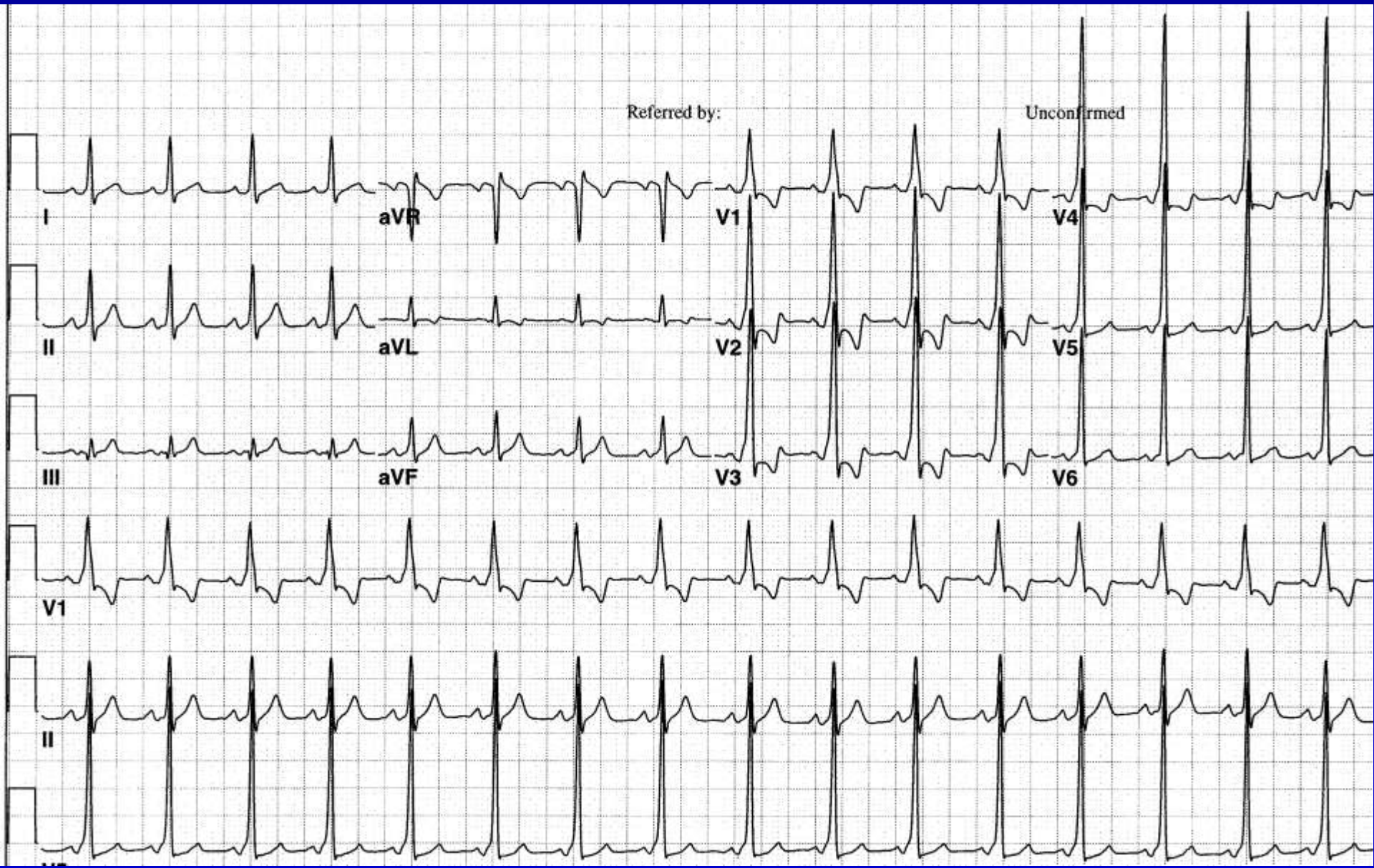
VAH0554

Left lateral or anterolateral with preexcited tachycardia (AF)

Atrial fib with long RR is low risk, high risk if RR<250 ms



NSR and WPW Pattern



WPW and Atrial Fibrillation

Course of Bypass Tract

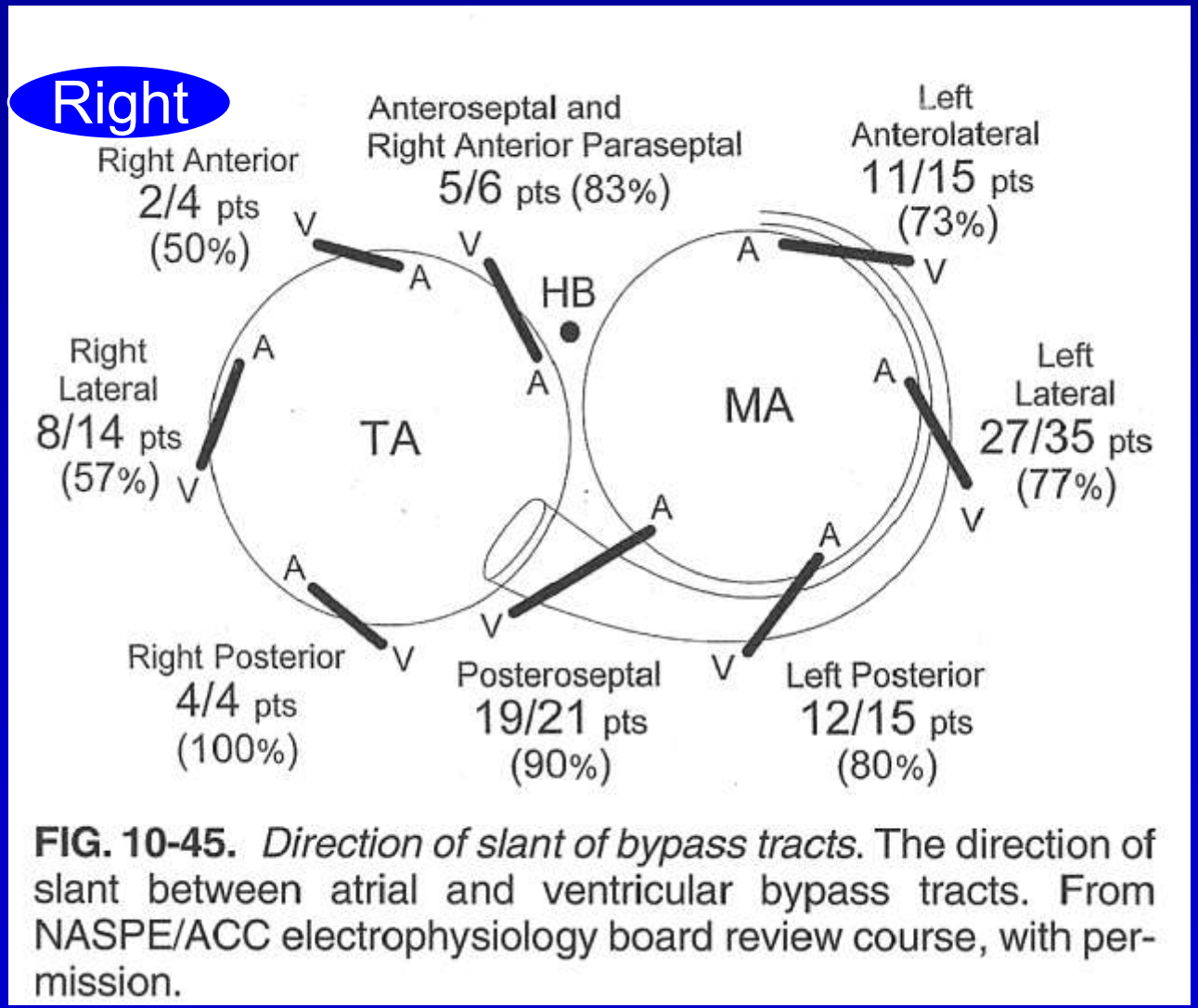
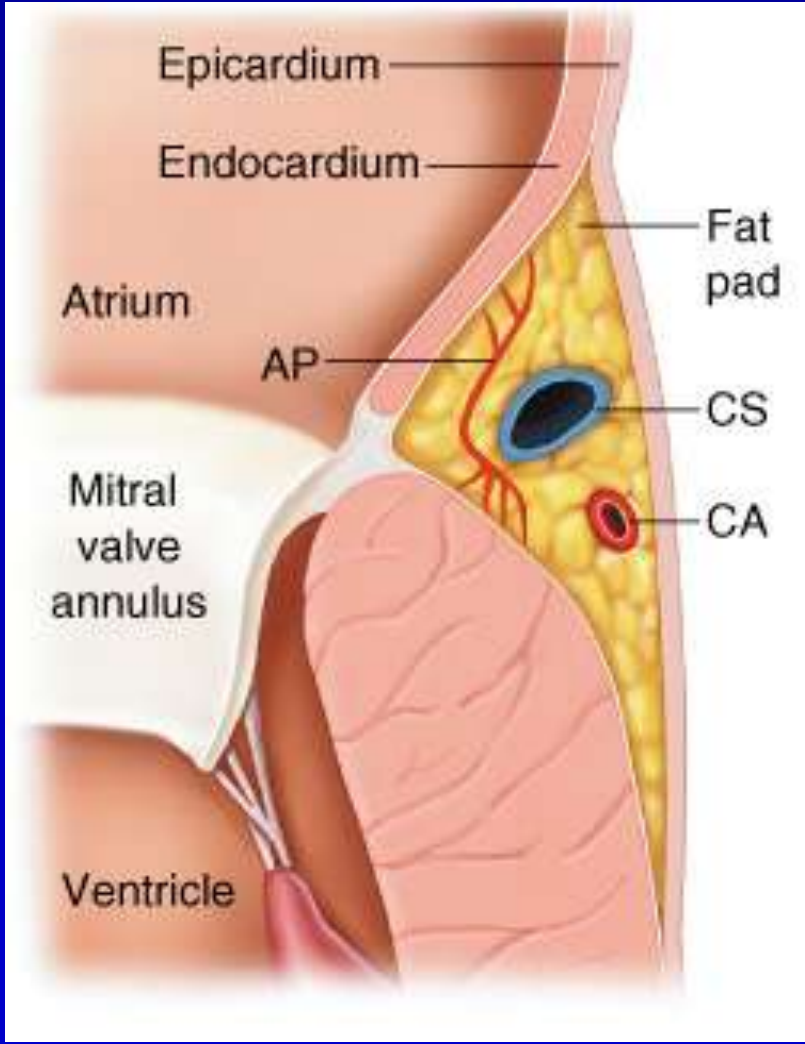


FIG. 10-45. *Direction of slant of bypass tracts.* The direction of slant between atrial and ventricular bypass tracts. From NASPE/ACC electrophysiology board review course, with permission.

Josephson ME. 2002, p. 358.

Ventricular Pre-excitation

- Conduction is usually rate-independent like ventricular muscle
- Rate-dependent (decremental conduction) conduction to some degree has been found in about 7-8% (“PJRT”)
  - Antegrade decremental conduction more in right side
  - Retrograde decremental conduction anywhere



Zipes and Jalife, 5th ed, 2009, p. 605-613; Braunwald, 2008, p. 819. *Peters NS et al. Eur Heart J. 1994;15:981.

PJRT: Permanent Form of Junctional Reciprocating Tachycardia (Coumel)

The circuit involves 2 pathways with slow conduction, so giving a large "excitable gap"

- Incessant or nearly so, esp. seen in young
- Almost all cases due to retrograde conduction over accessory pathway, so better term is PAVRT, and accessory pathway has decremental retrograde conduction
- P waves are usually broad, inverted in 2, 3, and aVF
- RP longer than PR ("Long RP tachycardia")

Zipes and Jalife, 4th ed, 2004, p. 869-878

PJRT: Permanent Form of Junctional Reciprocating Tachycardia

- Initiation of arrhythmia is with sinus beat, not PAC
- Rate of arrhythmia is sensitive to autonomic tone and physical activity with modulation of both RP and PR intervals
- Transient termination of arrhythmia through block in retrograde limb (no P wave)
- Retrograde limb sensitive to β-blockade, vagal maneuvers and calcium blockade, but arrhythmia is often refractory to medication

Zipes and Jalife, 4th ed, 2004, p. 869-878

History of WPW Ablation

- 1967 – temporary ablation of pathway at surgery using procaine injection
- 1968 – ablation of pathway at surgery (Duke: Will Sealy, John Boineau, Galen Wagner, Andrew Wallace)
- 1984 – ablation of pathway with 200J DC shock
- 1989 – ablation of pathway with radiofrequency current
- 2001 – ablation of pathway with cryotherapy

Assessment of Risk in WPW

- Risk of SCD is higher in some subgroups
  - RR interval in AFib <250 msec
  - History of symptomatic tachycardia
  - Multiple accessory pathways
  - Ebstein's anomaly
  - Familial WPW (rare)
- Risk of SCD is lower in some subgroups
  - Intermittent absence of delta wave
  - Asymptomatic ECG abnormality in pt >40 yo

Therapy for WPW Pattern on ECG (not syndrome)

- Asymptomatic patients with incidental preexcitation – no further eval or mgmt UNLESS … poss high-risk occupation such as bus driver, pilot, scuba diver, police, military, competitive athletics

Acute therapy for WPW

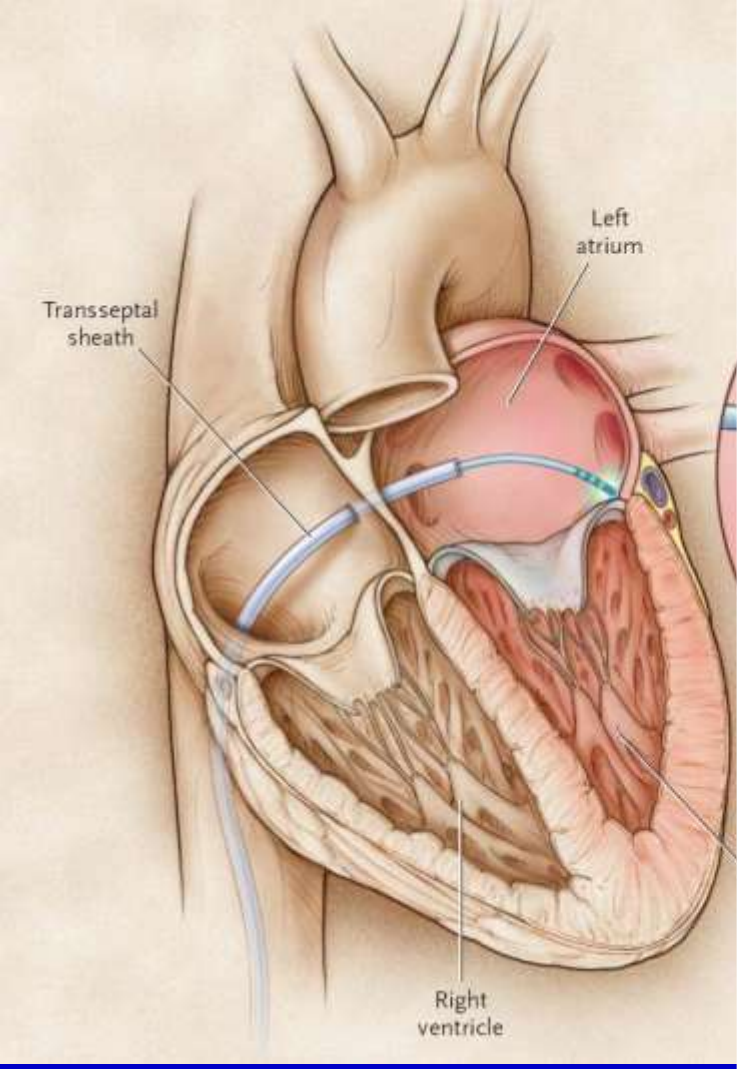
- Acute therapy for stable orthodromic AVRT: IV adenosine is highly effective (caution, it may result in atrial fibrillation in up to 12%) - - if asthmatic, IV calcium blocker
- Acute therapy for stable “preexcited tachycardia” (incl. antidromic AVRT, AT, Aflutter, AFib, AVNRT): IV procainamide or ibutilide
- Acute therapy for unstable tachycardia: DC cardioversion

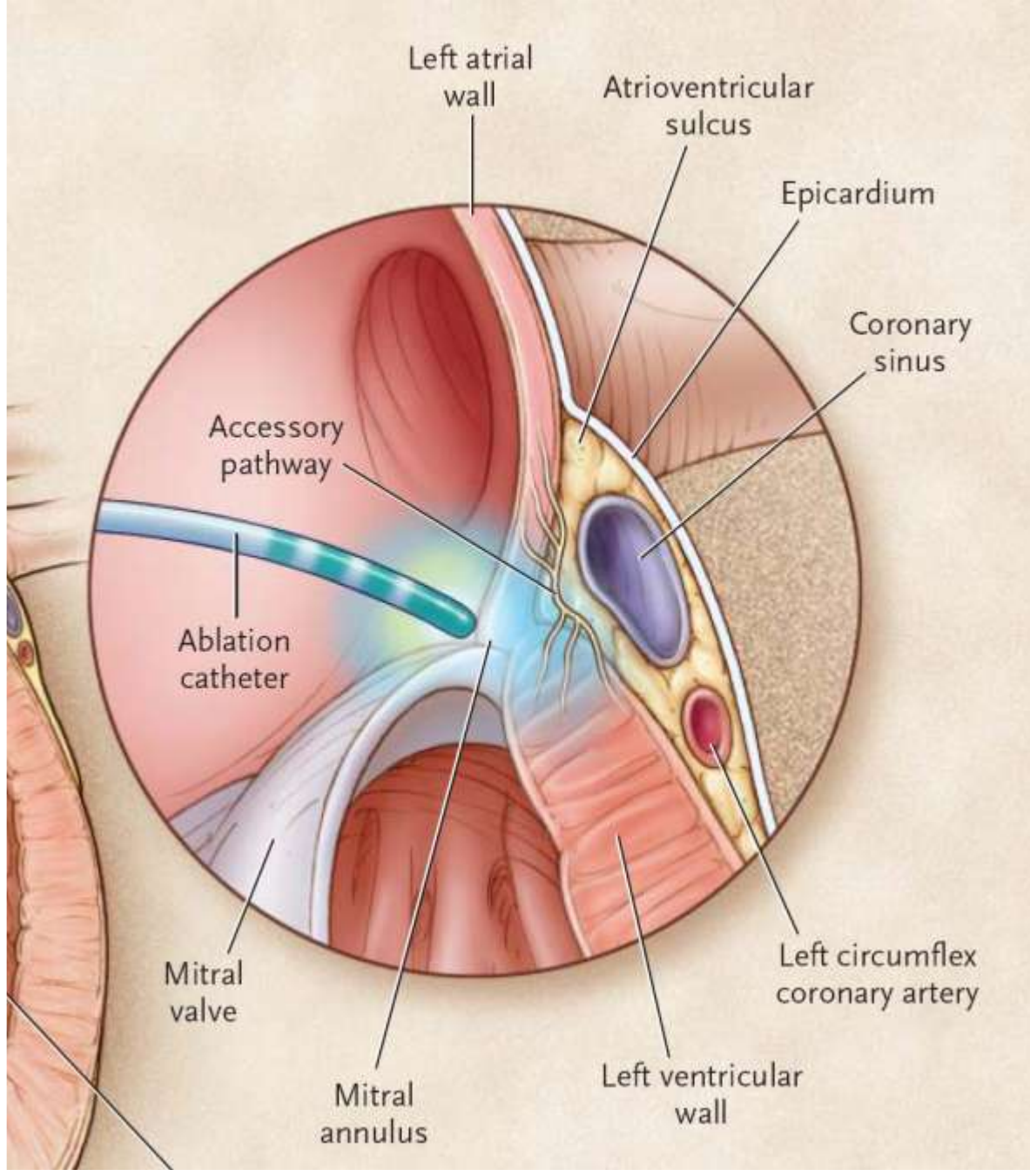
Chronic therapy for WPW

- Infrequent well-tolerated episodes possible to use “pill-in-the-pocket” approach with AV nodal blocking agent
- Recurrent episodes, medical therapy with beta-blocker or calcium blocker VS ablation
- If persistent episodes, may add propafenone, flecainide, sotalol, or amiodarone to block accessory pathway

Catheter Ablation for WPW

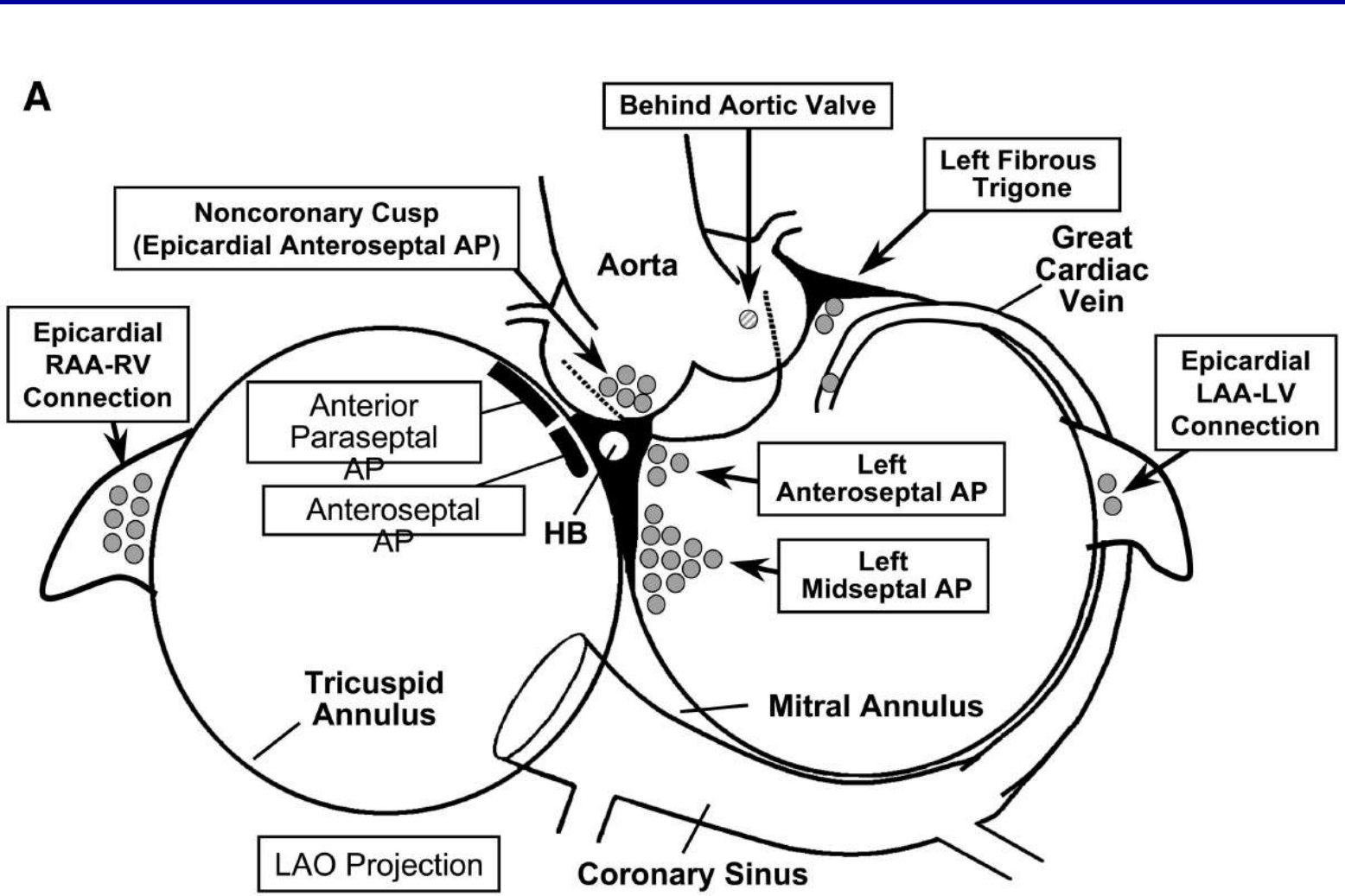
- Radiofrequency for most, cryoablation for tracts near the conduction system or near coronary arteries (in coronary sinus)
- Cryoablation allows for “ice mapping”, technique of temporary freezing to evaluate result – if result is desirable, can perform permanent freeze





Lerman BB et al. NEJM. 2003;349:1787.

Unusual Pathway Locations



Nakagawa H, Jackman WM. Circulation. 116:2465-2478; 2007.

Catheter Ablation for WPW

- Success rate 98%
- Repeat procedure rate 2.2%
- Serious complication rate 0.6%
  - Tamponade, AV block, coronary artery injury, retroperitoneal bleed, stroke
- Mortality 0.02%
- (Overall annual SCD risk in WPW is 0.05-0.5%)

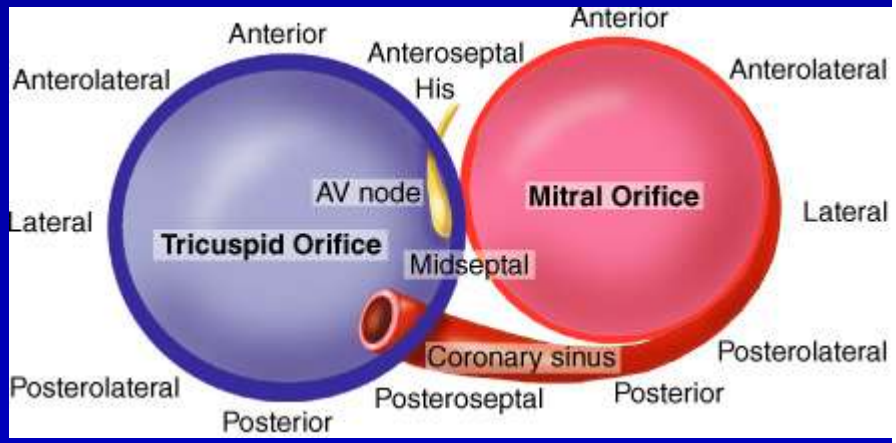
Indications for Catheter Ablation for WPW

- WPW syndrome with symptomatic arrhythmias, well tolerated (I)
- WPW syndrome with AF and rapid-conduction or poorly tolerated AVRT (I)
- AVRT, poorly tolerated, no pre-excitation (I)
- Single or infrequent AVRT episodes, no pre-excitation (IIa)
- Asymptomatic pre-excitation (IIa)

Future in WPW

- Progress in genetic associations and pathogenesis/embryogenesis
- Incremental improvements in ablation technology
  - High intensity focused ultrasound

Pathway Locations



Caveat: different authors use different orientations of the mitral and tricuspid orifices in their illustrations

Surawicz, Chou, 2001, p. 478
Braunwald, 2005, p. 740

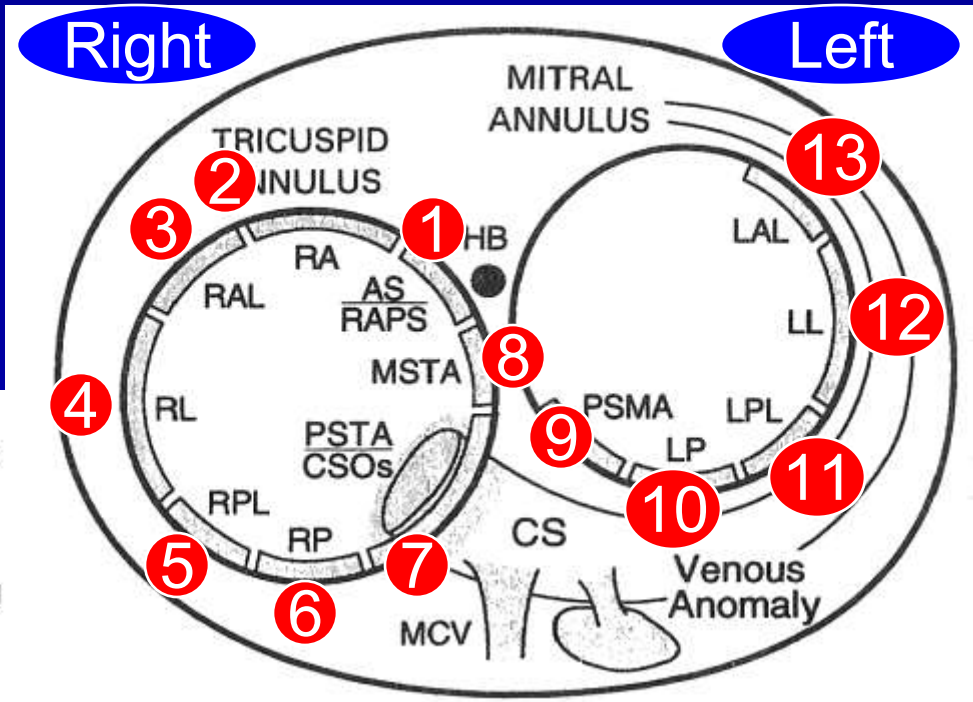
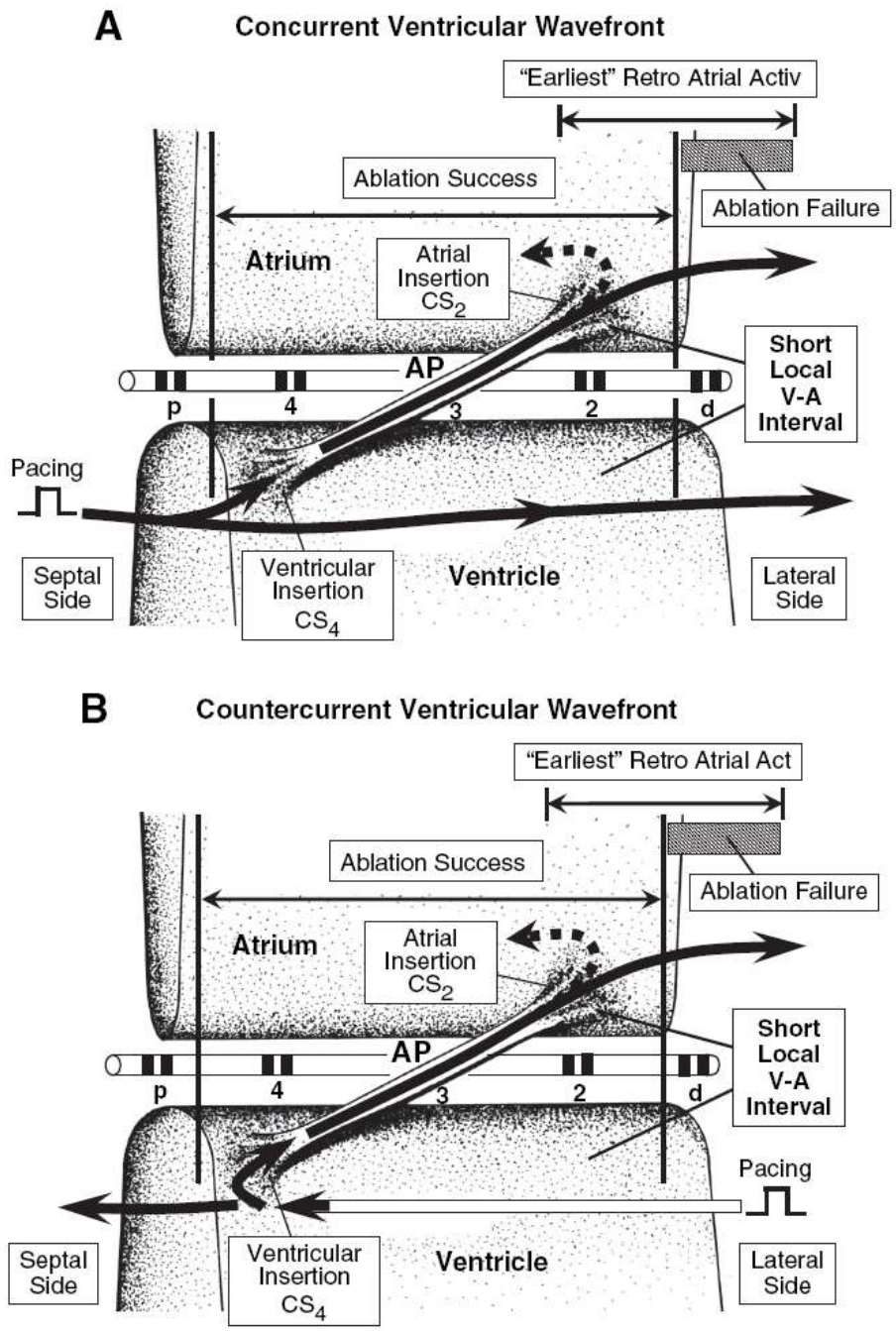


Figure 20–19. The heart as viewed in the left anterior oblique projection. Nomenclature used to describe accessory pathway locations. RA = right anterior; RAL = right anterolateral; RL = right lateral; RPL = right posterolateral; RP = right posterior; PSTA = posteroseptal tricuspid annulus; CSOs = coronary sinus ostium; MSTA = midseptal tricuspid annulus; AS = anteroseptal; RAPS = right anterior paraseptal; MCV = middle cardiac vein (coronary vein); CS = coronary sinus venous anomaly (coronary sinus diverticulum); PSMA = posteroseptal mitral annulus; LP = left posterior; LPL = left posterolateral; LL = left lateral; LAL = left anterolateral; HB = His bundle. (From Arruda MS, McClelland JH, Wang X, et al: Development and validation of an ECG algorithm for identifying accessory pathway ablation site in Wolff-Parkinson-White syndrome. J Cardiovasc Electrophysiol 9:2, 1998, by permission.)

Ablation of Oblique Pathway



Nakagawa H and Jackman WM. Circulation. 116:2465-2478; 2007.

Locations of Accessory Pathways

(Finer subdivisions are also used)

- Right anteroseptal (less common)
- Right free wall (third most common, 10-20%)
- Posteroseptal (second most common, 20-30%)
- Mid-septal (between His bundle and coronary sinus, less common)
- **Left free wall** (most common, 50-60%)
- Multiple in 5-20% of patients
  - Particularly posteroseptal and right free wall: consider Ebstein's
  - More in patients resuscitated from VF

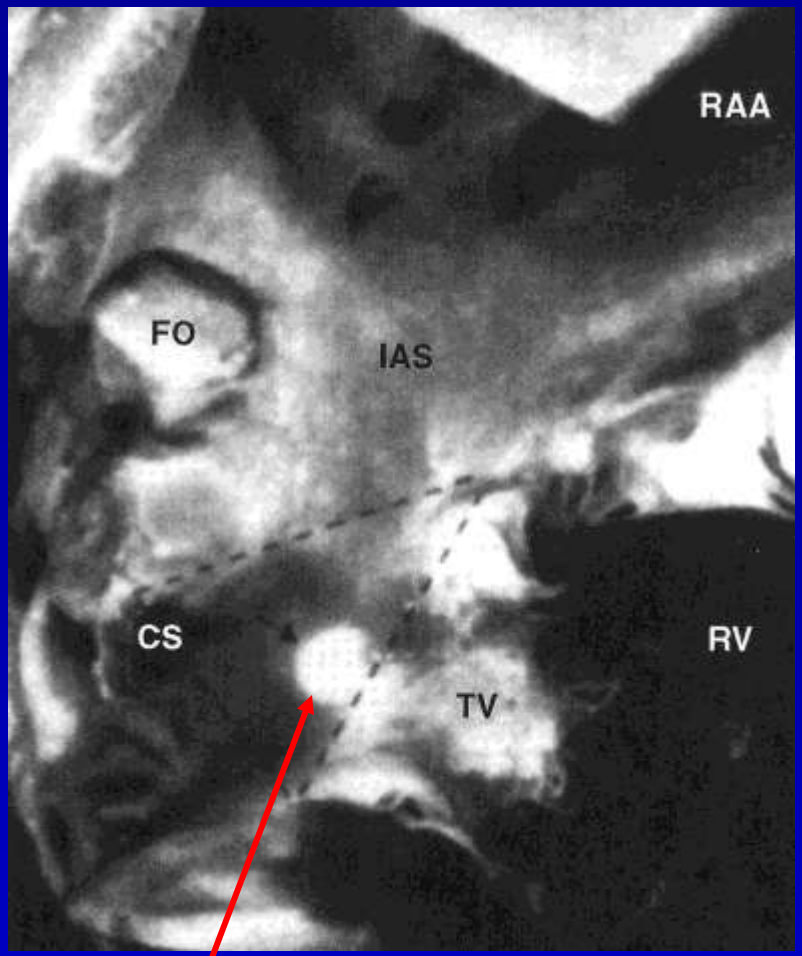
Zipes and Jalife, 4th ed, 2004, p. 869-878

Names of Fibers

- Kent: AV connection
- James: atrium to distal or compact AVN
- Brechenmacher: atrium to His bundle
- Mahaim: His to ventricle (commonly used for atriofascicular fiber, original description was nodofascicular connection which is much less common than atriofascicular, e.g., atrium to right bundle branch along the lateral tricuspid annulus only capable of anterograde connection)

Podrid and Kowey. Cardiac Arrhythmias: Mechanisms, diagnosis, and therapy. 2001. Ch. 17 “Tachycardias in WPW;” by Marinchak RA and Rials SJ, p. 517.
Zipes DP and Jalife J. Cardiac Electrophysiology: From Cell to Bedside; 2004. Ch. 58, “Atrioventricular Reentry and Variants” by Knight BP and Morady F, p. 528ff. And Ch. 94, “Wolff-Parkinson-White Syndrome;” by Prystowsky E et al, p. 869ff.

Types of Fibers



Site of prior successful ablation of AVNRT

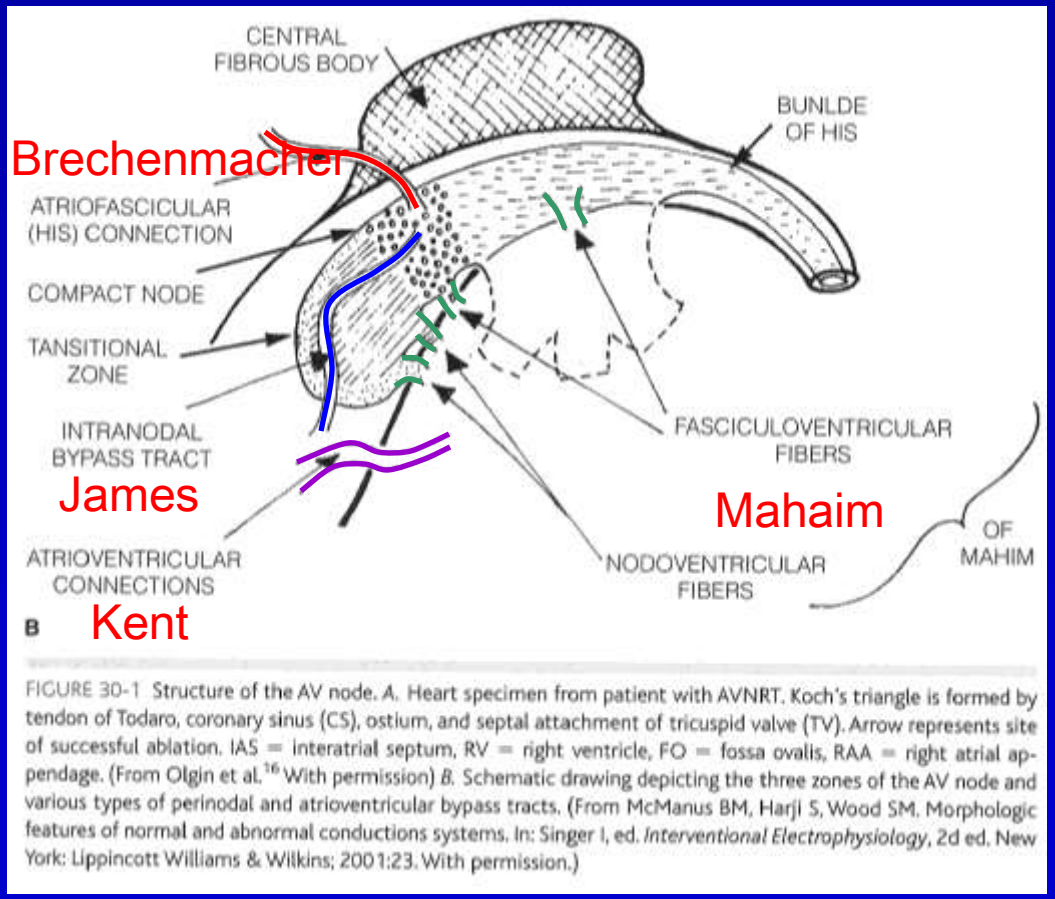
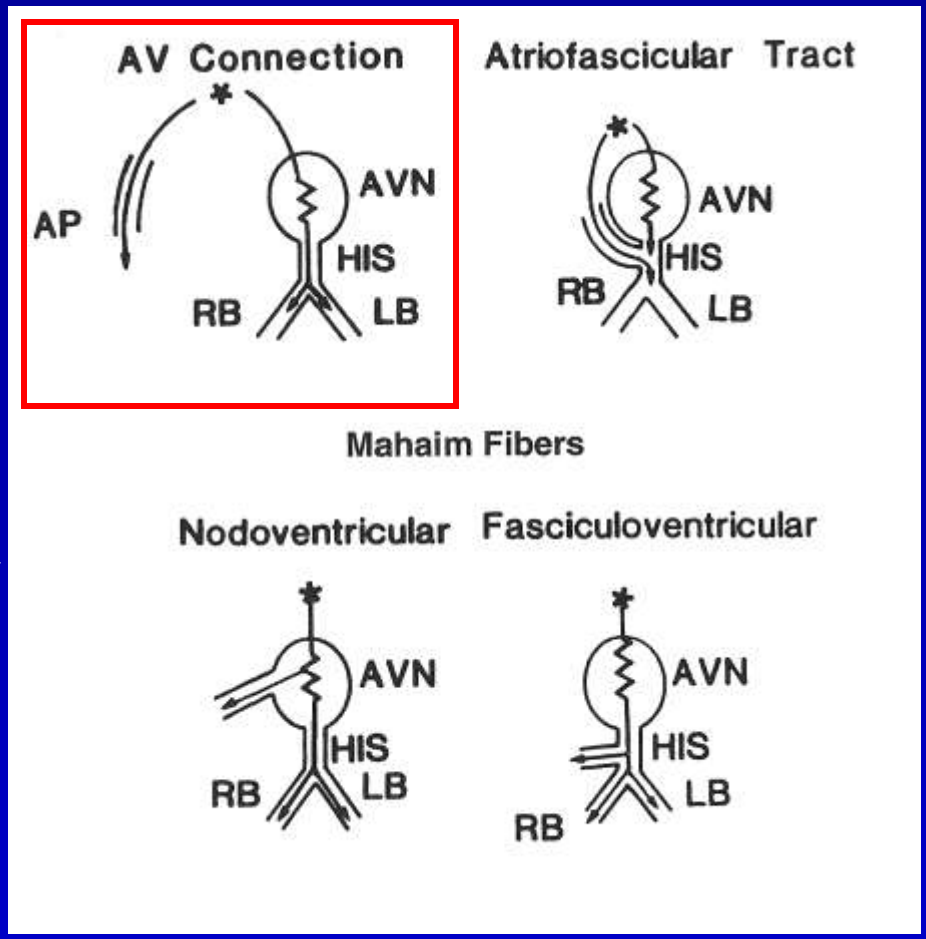


FIGURE 30-1 Structure of the AV node. *A.* Heart specimen from patient with AVNRT. Koch's triangle is formed by tendon of Todaro, coronary sinus (CS), ostium, and septal attachment of tricuspid valve (TV). Arrow represents site of successful ablation. IAS = interatrial septum, RV = right ventricle, FO = fossa ovalis, RAA = right atrial appendage. (From Olgin et al.[16] With permission) *B.* Schematic drawing depicting the three zones of the AV node and various types of perinodal and atrioventricular bypass tracts. (From McManus BM, Harji S, Wood SM. Morphologic features of normal and abnormal conductions systems. In: Singer I, ed. *Interventional Electrophysiology*, 2d ed. New York: Lippincott Williams & Wilkins; 2001:23. With permission.)

Hurst, 11th ed. P. 856.

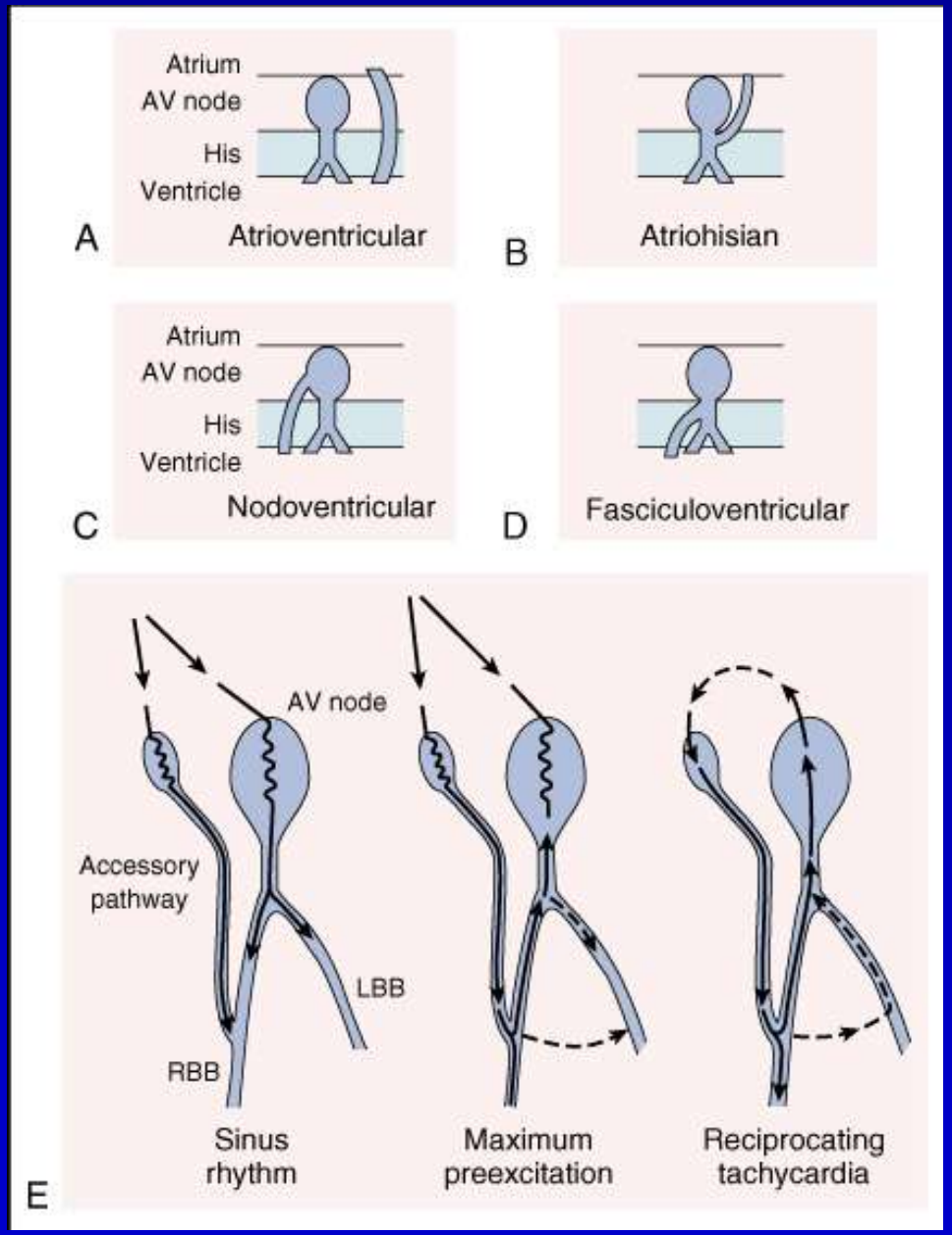
Types of Ventricular Pre-Excitation

- Atrioventricular pathway (Kent bundle) – most common
- Mahaim fibers
  - Atriofascicular (Brechenmacher tract is to His bundle), antegrade only and decremental so reciprocating tachycardia is LBBB
  - *Nodoventricular (and nodofascicular) – clinical significance is controversial, reciprocating tachycardia does not require atrium (so can dissociate)
  - Fasciculoventricular - has not been demonstrated to cause



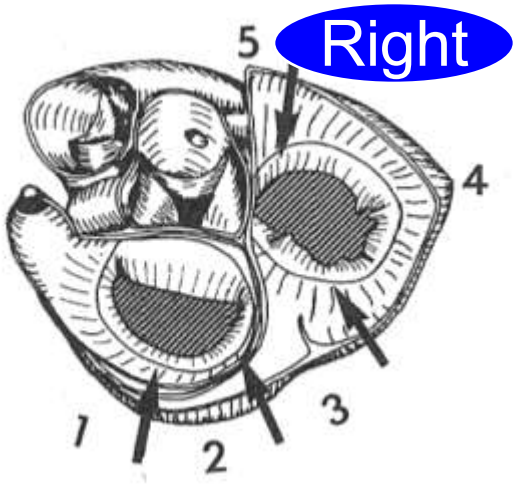
Types of Ventricular Pre-Excitation

A. Atrioventricular pathway (Kent bundle) – most common
B. Atriohisian is very uncommon, might give LGL, unproved (but atriofascicular does exist and gives preexcitation)
C. Nodoventricular, original concept
D. Fasciculoventricular, not thought to be important in genesis of arrhythmia
E. Current concept of nodoventricular – accessory pathway with AV nodal properties



Braunwald, Ch. 32, "Specific Arrhythmias: Diagnosis and Treatment", Olgin JE and Zipes DP. p. 830, 2005

Localization of Bypass Tract



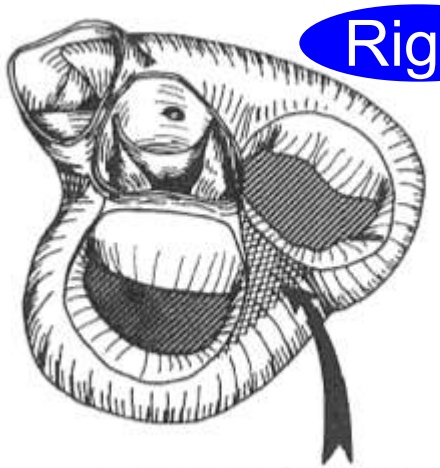


FIG. 10-42. *Schema of bypass tract locations used for ECG analysis.* On the left the heart is opened at the midatrial level, and on the right the atria have been removed. The regions we find useful for ECG classification of bypass tracts are shown on the left. Region 1 is left lateral, region 2 is left posterior free wall, region 3 is posterior septal, region 4 is right free wall, and region 5 is anterior septal. The area between 3 and 5 along the tricuspid valve incorporates what are now referred to as mid-septal pathways. See text for discussion.

Josephson ME. 2002, p. 356

Preexcitation Syndromes

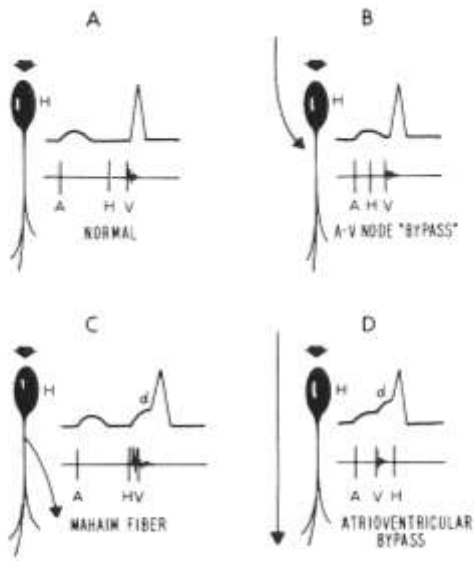


Fig. 1. Types of accessory pathways. The electrocardiogram, His bundle electrogram, and schematic of the conducting pathways associated with a normal conduction system (A), an atrioventricular node "bypass" (B), a Mahaim fiber (C), and a complete atrioventricular bypass (D) are shown. A, atrial electrogram; H, His bundle electrogram; V, ventricular electrogram; d, delta wave. (Reproduced by permission.[18])

Table 1. Spectrum of Preexcitation Syndromes

Syndrome	No. Patients
Accessory atrioventricular connections (WPW)	163
With associated EAVN*	20
AP conducts only retrograde	12
EAVN (LGL or variant) alone	11
Nodoventricular fibers	2
Fasciculoventricular fibers	6
EAVN plus fasciculoventricular fiber (mimicking WPW)	4

*EAVN, enhanced atrioventricular node conduction.

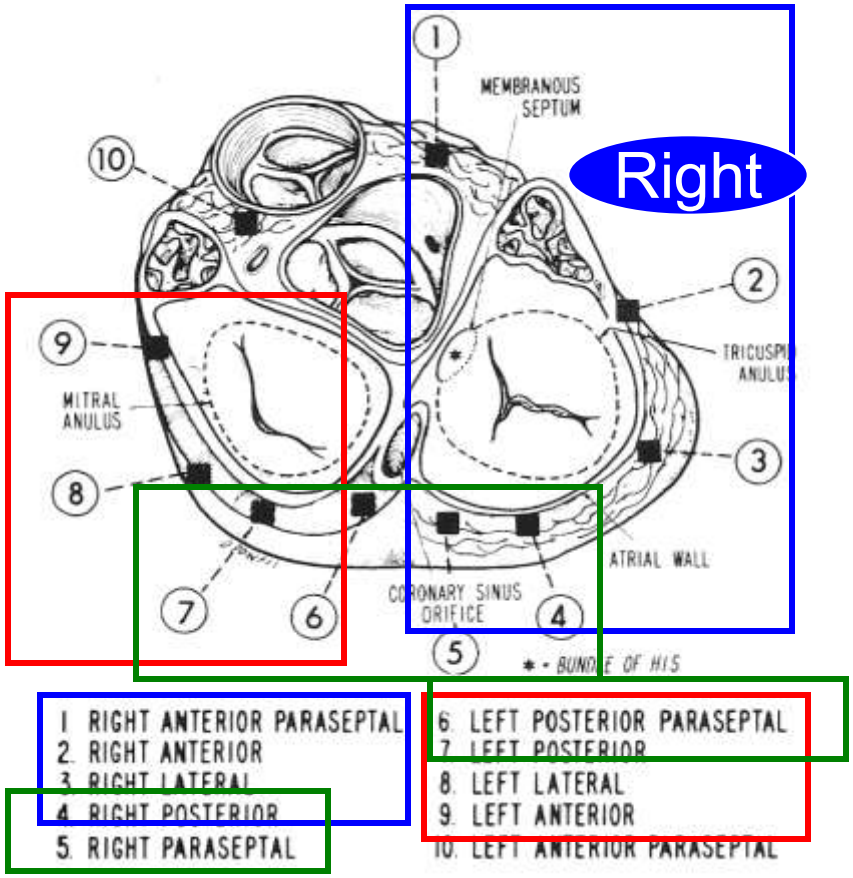
Table 2. Proposed Terminology for Anatomic Substrates of the Preexcitation Syndromes

Proposed Terminology	Previous Terminology
Accessory AV connection	Kent bundle (in septum also called Paladino tract)
Atriofascicular bypass tract	Atrio-Hisian fiber
Intranodal bypass tract*	James fiber
Nodoventricular connection	Mahaim fiber
Fasciculoventricular connection	Mahaim fiber

*Enhanced AV conduction through the AV node may be equally well explained by an AV nodal malformation or functional states of conduction unaccompanied by abnormal anatomic substrates.

Gallagher JJ, Pritchett ELC, Sealy WC, Kasell J, Wallace AG. "The preexcitation syndromes," Prog Cardiovasc Dis. 1978;20(4):285-327.

Localization of Bypass Tract



DELTA WAVE POLARITY

	I	II	III	AVR	AVL	AVF	V_1	V_2	V_3	V_4	V_5	V_6
①	+	+	+(±)	-	±(+)	+	±	±	+(±)	+	+	+
②	+	+	-(±)	-	+(±)	±(-)	±	+(±)	+(±)	+	+	+
③	+	±(-)	-	-	+	-(±)	±	±	±	+	+	+
④	+	-	-	-	+	-	±(+)	±	+	+	+	+
⑤	+	-	-	-(+)	+	-	±	+	+	+	+	+
⑥	+	-	-	-	+	-	+	+	+	+	+	+
⑦	+	-	-	±(+)	+	-	+	+	+	+	+	-(±)
⑧	-(±)	±	±	±(+)	-(±)	±	+	+	+	+	-(±)	-(±)
⑨	-(±)	+	+	-	-(±)	+	+	+	+	+	+	+
⑩	+	+	+(±)	-	±	+	±(+)	+	+	+	+	+

± = Initial 40 msec delta wave isoelectric
+ = Initial 40 msec delta wave positive
- = Initial 40 msec delta wave negative

Fig. 12. Electrocardiographic classification of the Wolff–Parkinson–White syndrome. Ten representatives sites of epicardial preexcitation are depicted on a schematic cross-section of the ventricles at the level of the atrioventricular rings. The expected polarity of the delta wave resulting from preexcitation at these sites is indicated for each of the 12 standard ECG leads, based on analysis of the mean initial forces (40 msec) of ventricular depolarization in documented cases of single accessory pathways with no associated anomalies.

Gallagher JJ, Pritchett ELC, Sealy WC, Kasell J, Wallace AG. "The preexcitation syndromes," Prog Cardiovasc Dis. 1978;20(4):285-327.

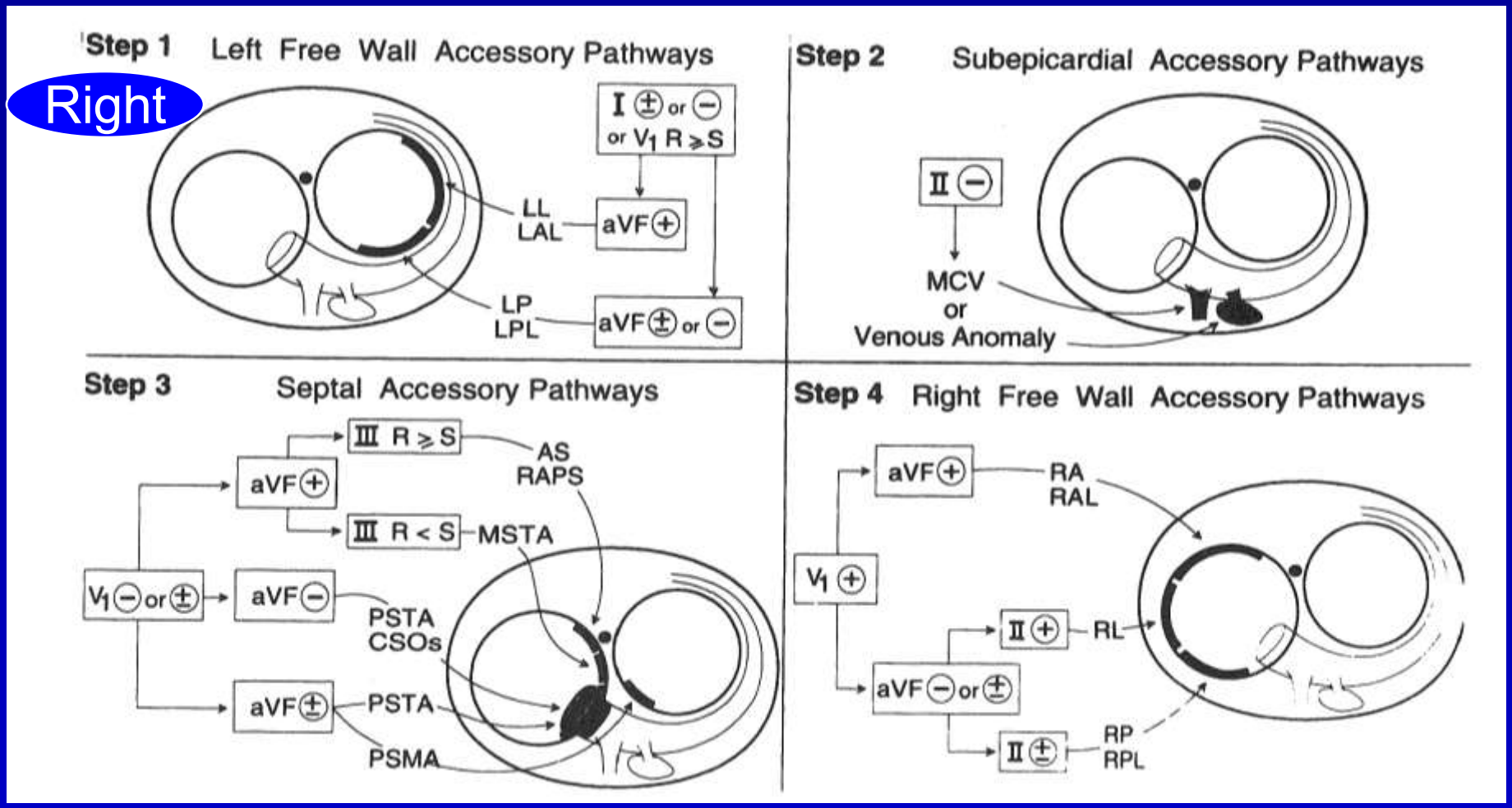
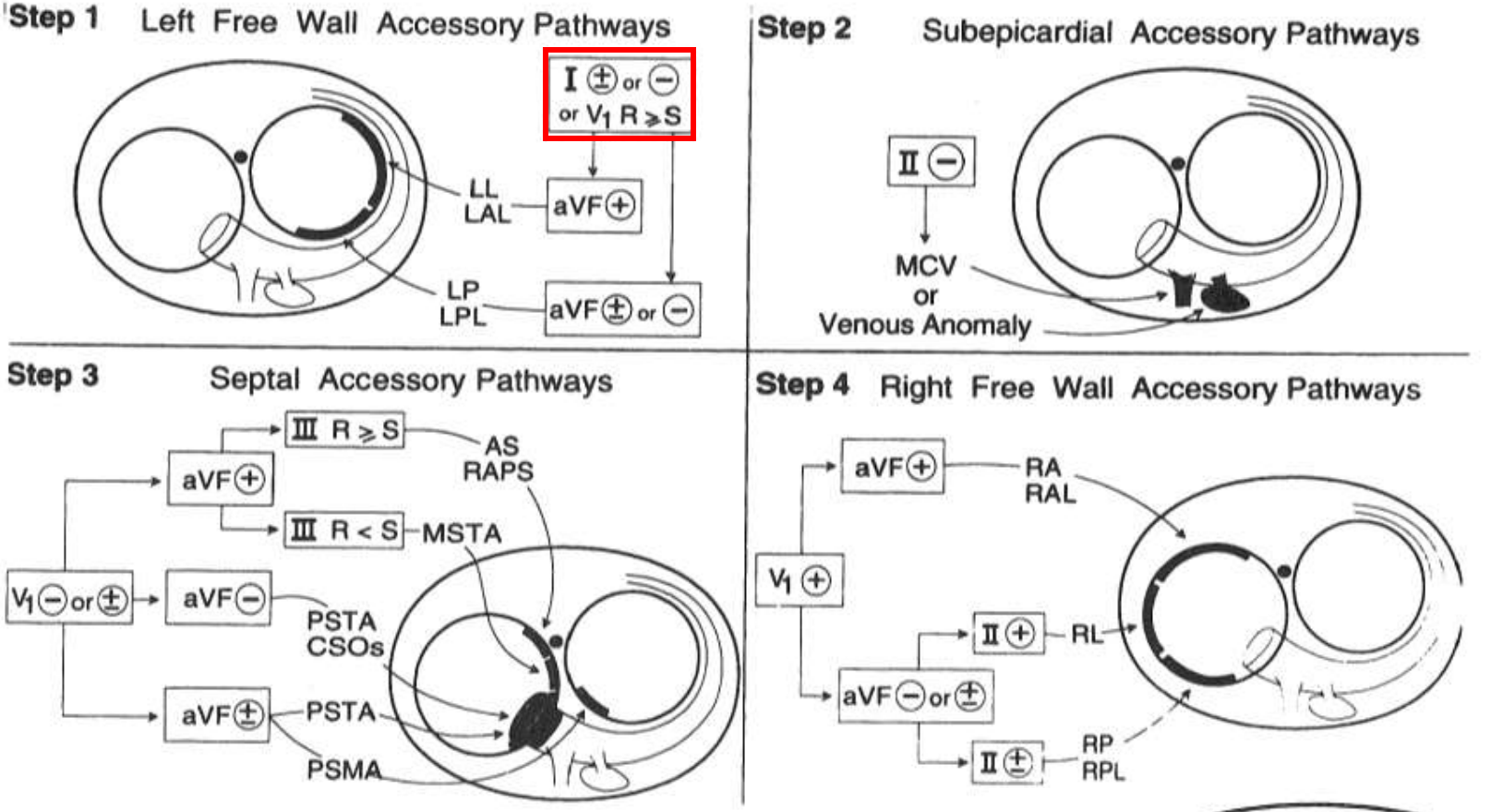
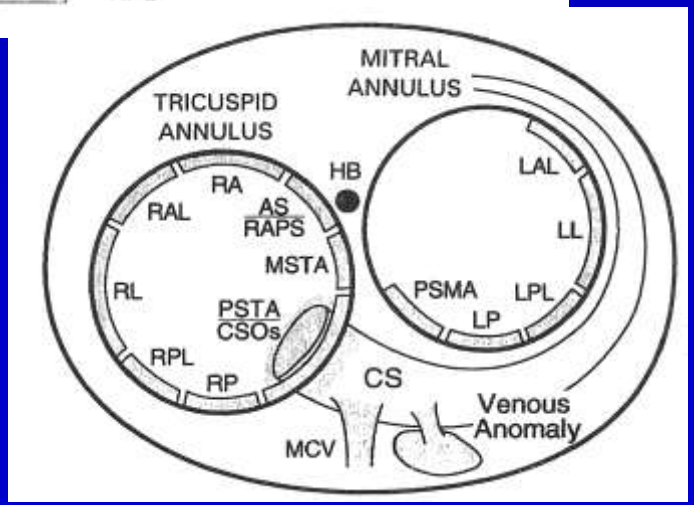


Figure 20–20. Stepwise electrocardiographic algorithm for predicting accessory pathway location. Abbreviations as in Figure 20–19. See text for explanation. (From Arruda MS, McClelland JH, Wang X, et al: Development and validation of an ECG algorithm for identifying accessory pathway ablation site in Wolff-Parkinson-White syndrome. J Cardiovasc Electrophysiol 9:2, 1998, with permission.)

Surawicz, Chou, 2001, p. 480

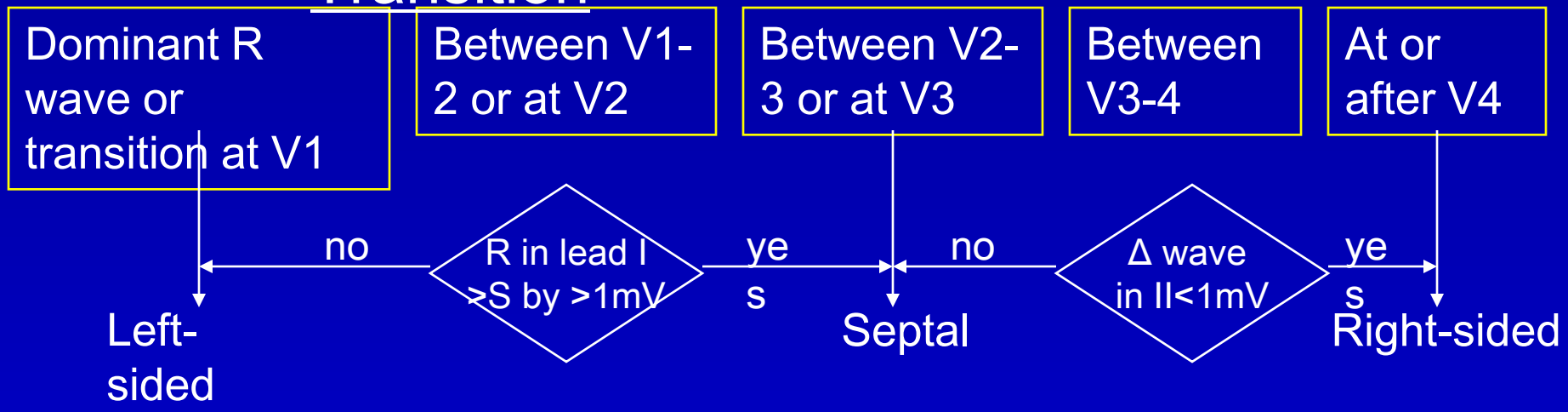




Surawicz, Chou, 2001, p. 478, 480

Localization of Pathway

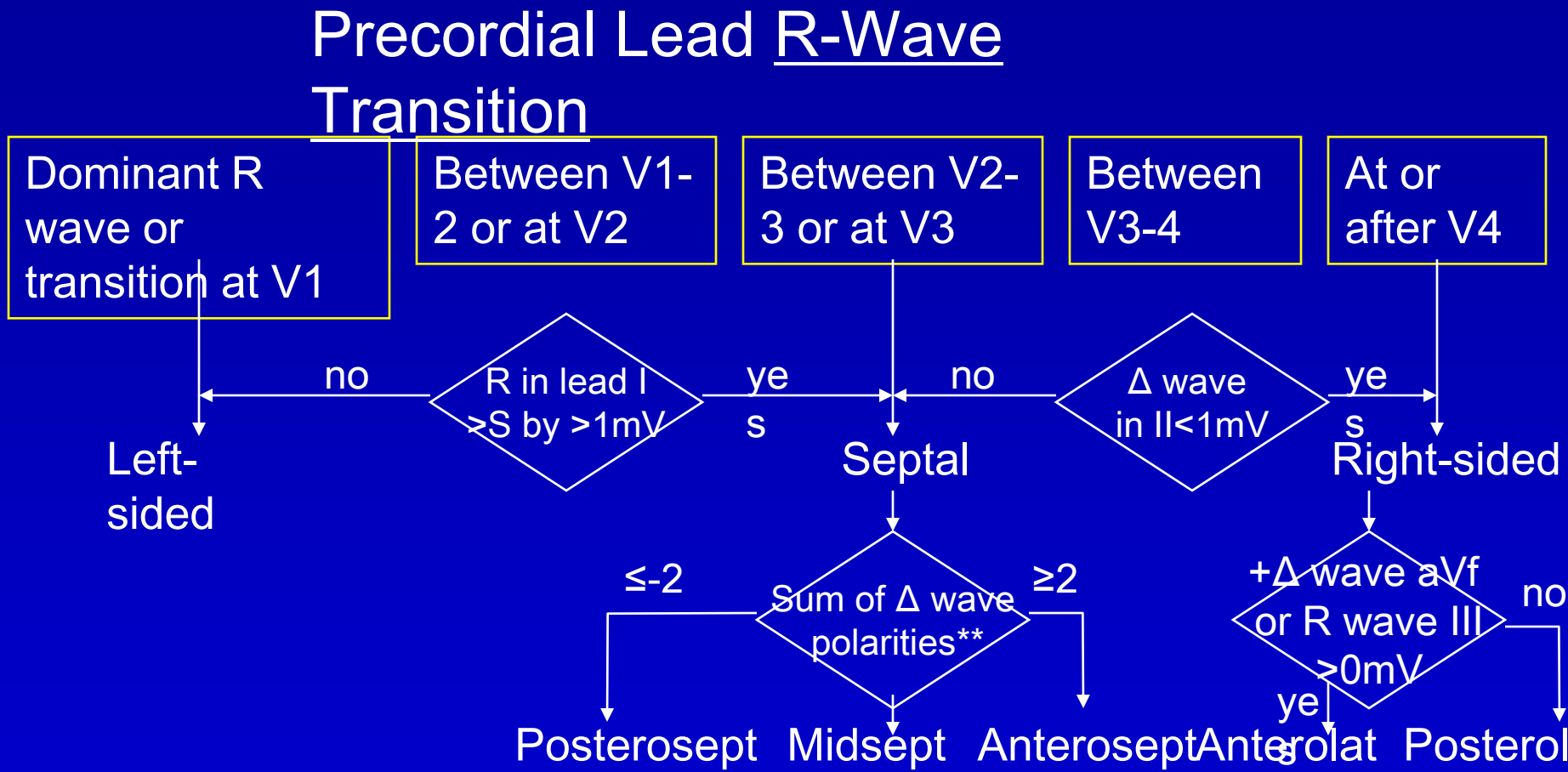
Precordial Lead R-Wave Transition*



*If the R/S ratio in any lead is nearly 1, the transition is at that lead. If R/S is <1 in one lead and >1 in the following lead, then the transition is between those leads.

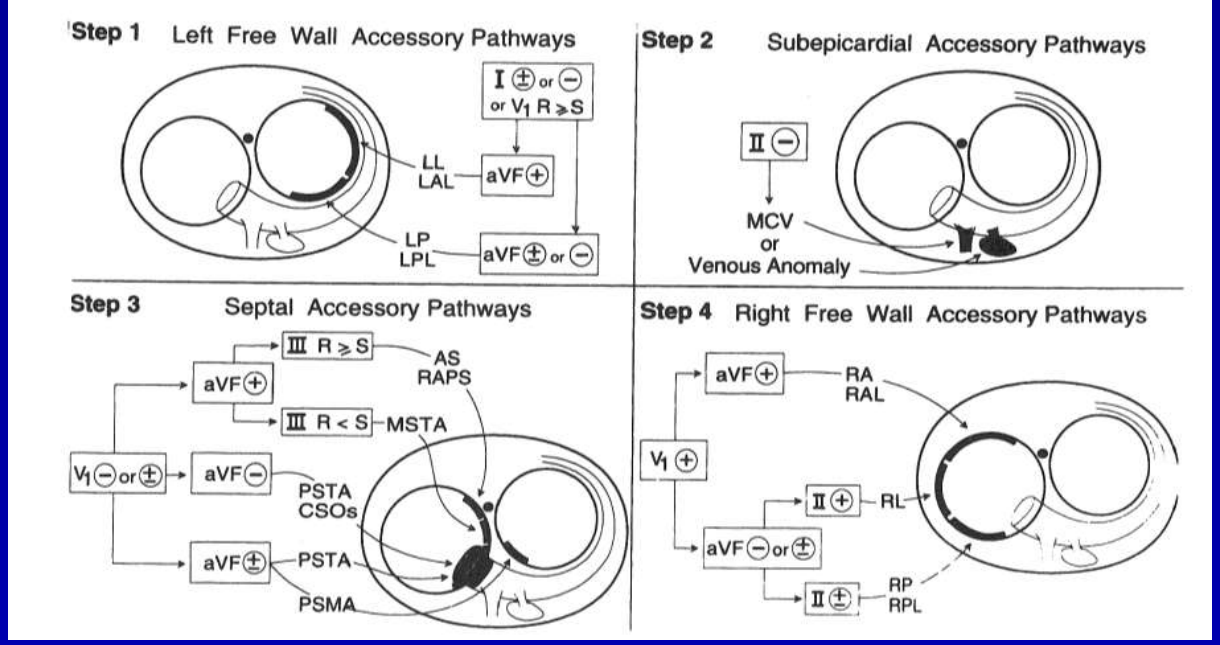
Zipes and Jalife, 3rd ed, 2000. Ch. 95, p. 1081

Localization of Pathway



** For separation of septal pathways, the sum of the delta wave polarities in the inferior leads is considered, where a positive delta wave = 1, a negative delta wave = -1, and an isoelectric delta wave = 0.

Zipes and Jalife, 3rd ed, 2000. Ch. 95, p. 1081



- Left free wall: negative delta wave in I, aVL, or V6 and “pseudo-RBBB” with Rs in V1
- Generally loss of a positive delta wave from leads 3 to aVF to 2 as the pathway location moves from anterior septal to posterior septal site around either AV ring
- Right anteroseptal (early ventricular activation near His bundle): positive delta wave in 2, 3, aVF, and low R/S in V1-V3 and late R wave transition
- Posteroseptal: negative or isoelectric delta waves in 2, 3, aVF and rapid R wave transition V1-V2
- Right free wall: positive delta wave in I and pseudo-LBBB
- For right-sided pathways, a positive delta wave occurs sequentially in V1 to V4 as the pathway location moves from anterior to posterior around the AV ring

Zipes and Jalife, 4th ed, 2004, p. 869-878; Surawicz, Chou, 2001, p. 480

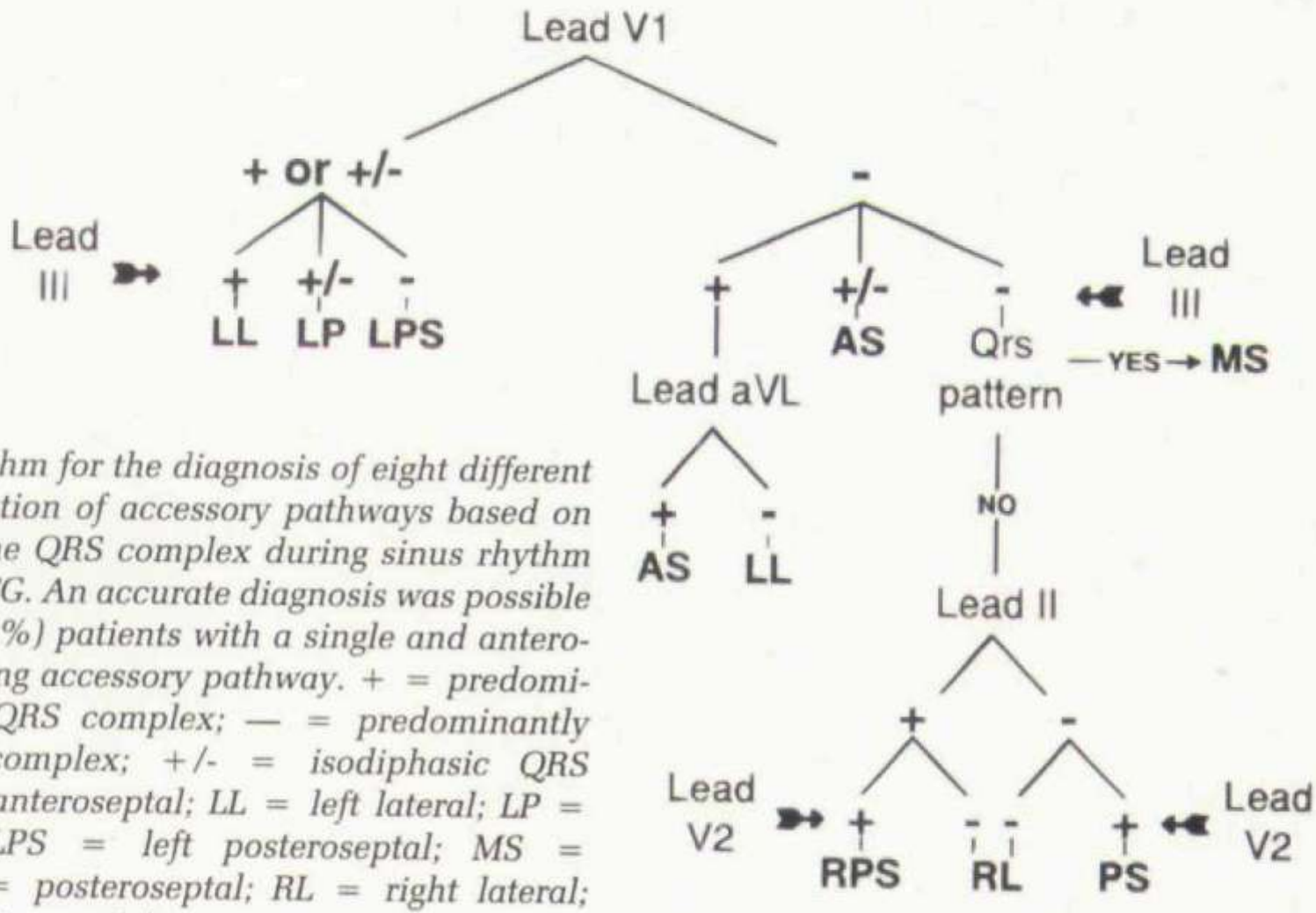


Figure 2. *Algorithm for the diagnosis of eight different sites of implantation of accessory pathways based on the polarity of the QRS complex during sinus rhythm on the surface ECG. An accurate diagnosis was possible in 128 of 140 (92%) patients with a single and antero-gradely conducting accessory pathway. + = predominantly positive QRS complex; — = predominantly negative QRS complex; +/- = isodiphasic QRS complex; AS = anteroseptal; LL = left lateral; LP = left posterior; LPS = left posteroseptal; MS = mid-septal; PS = posteroseptal; RL = right lateral; RPS = right posteroseptal.*

D'Avila A et al. PACE 1995;18:1615.

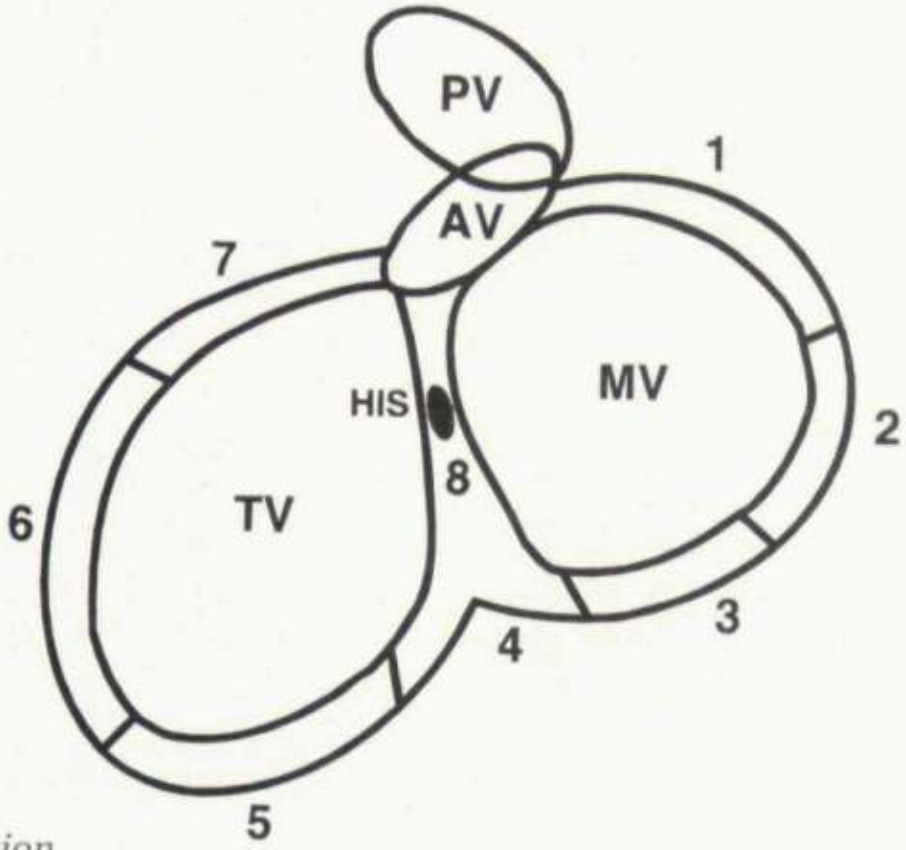
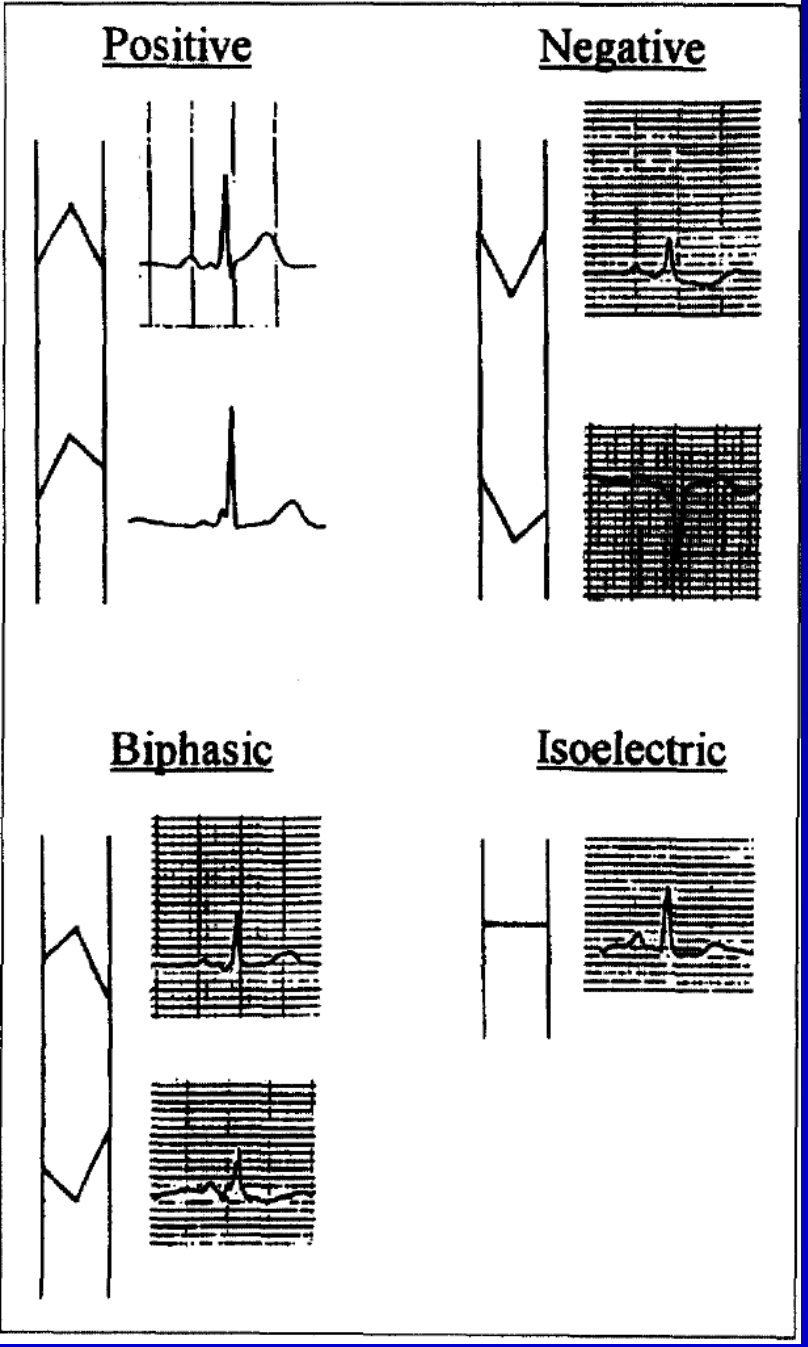


Figure 1. *Schematic drawings showing a cross-section of the atrioventricular ring on the 30° left anterior oblique projection. The possible anatomical localizations of the accessory pathways are shown. 1 = left lateral accessory pathway; 2 = left posterior accessory pathway; 3 = left paraseptal accessory pathway; 4 = posteroseptal accessory pathway; 5 = right paraseptal accessory pathway; 6 = right lateral accessory pathway; 7 = anteroseptal accessory pathway; 8 = midseptal accessory pathway; AV = aortic valve; MV = mitral valve; PV = pulmonic valve; TV = tricuspid valve.*

’Avila A et al. PACE 1995;18:1615.



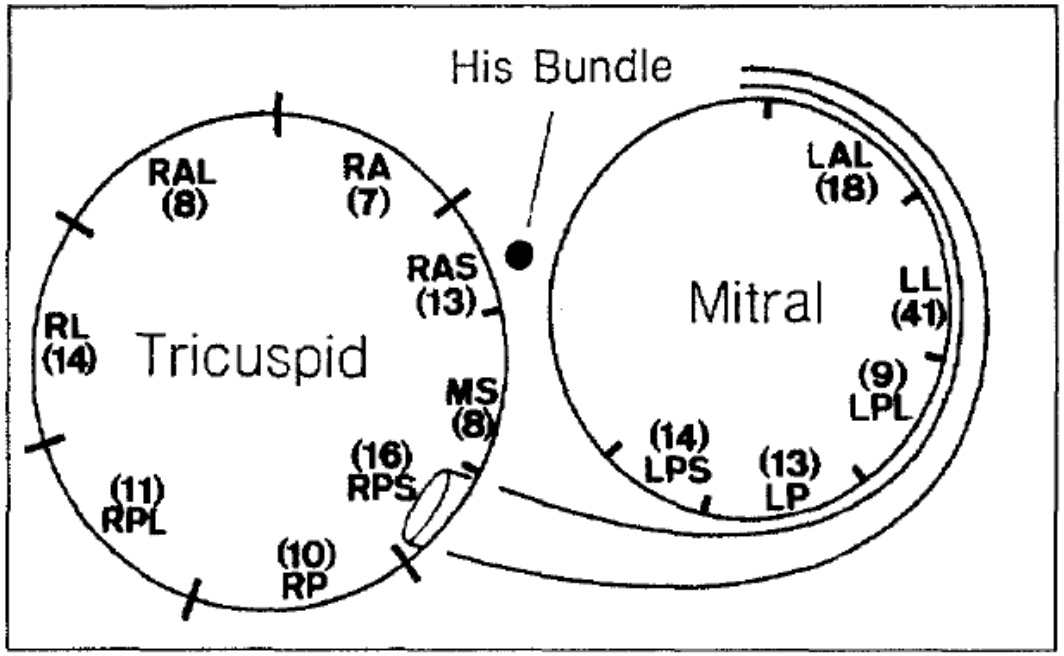
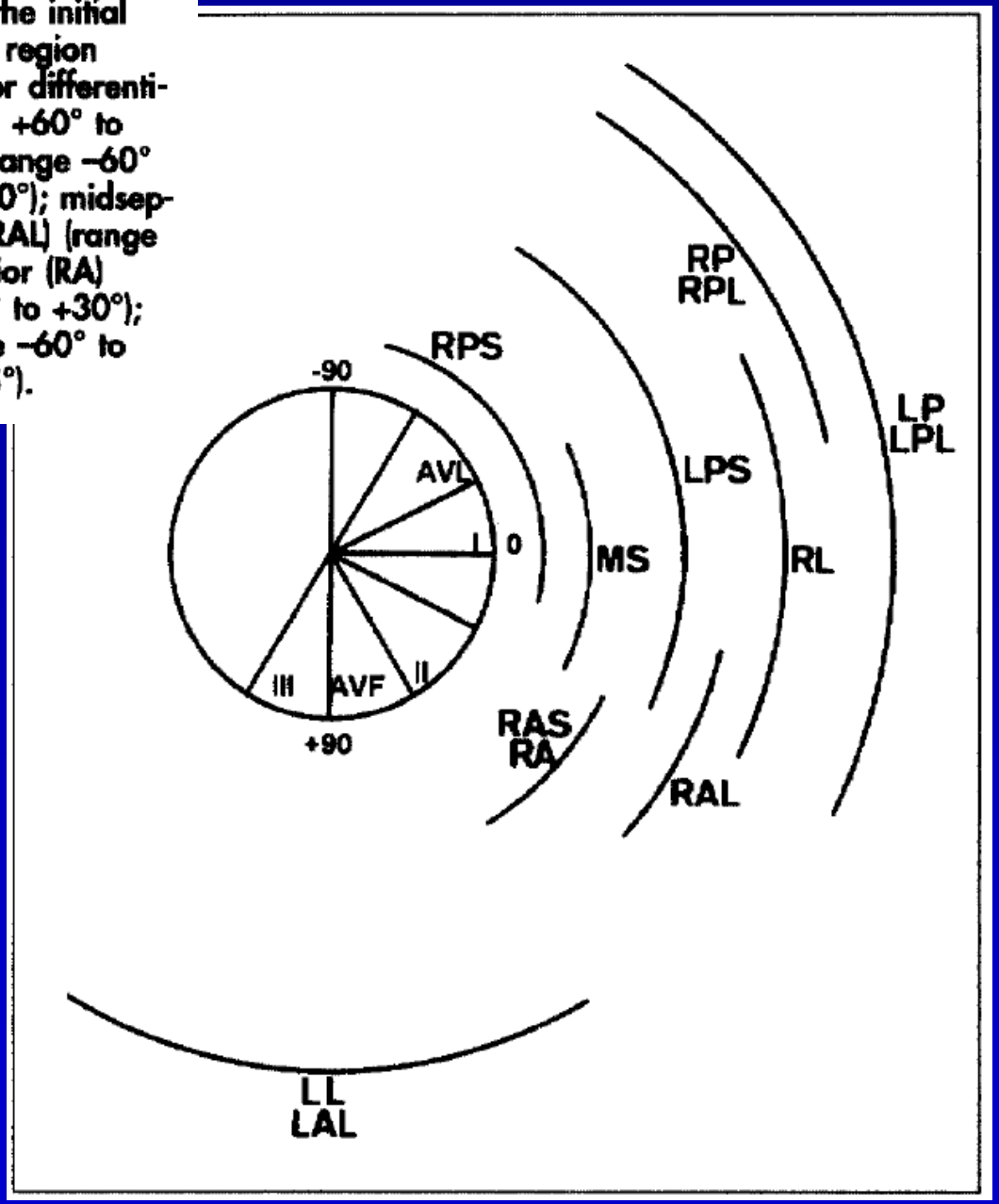


FIGURE 2. Schematic representation of the accessory pathway location in the best left anterior oblique projection, illustrating the division of the 13 regions. The coronary sinus and great cardiac vein are depicted encircling the mitral annulus, with the ostium demarcated by the venous phase of the left coronary arteriography, routinely performed in this laboratory before electrophysiologic study. The numbers of accessory pathways in each location of the 182 patients are shown in parentheses. LAL = left anterolateral; LL = left lateral; LP = left posterior; LPL = left posterolateral; LPS = left posteroseptal; MS = midseptal; RA = right anterior; RAL = right anterolateral; RAS = right anteroseptal; RL = right lateral; RP = right posterior; RPL = right posterolateral; RPS = right posteroseptal.

Chiang CE et al. Am J Cardiol 1995;76:40.

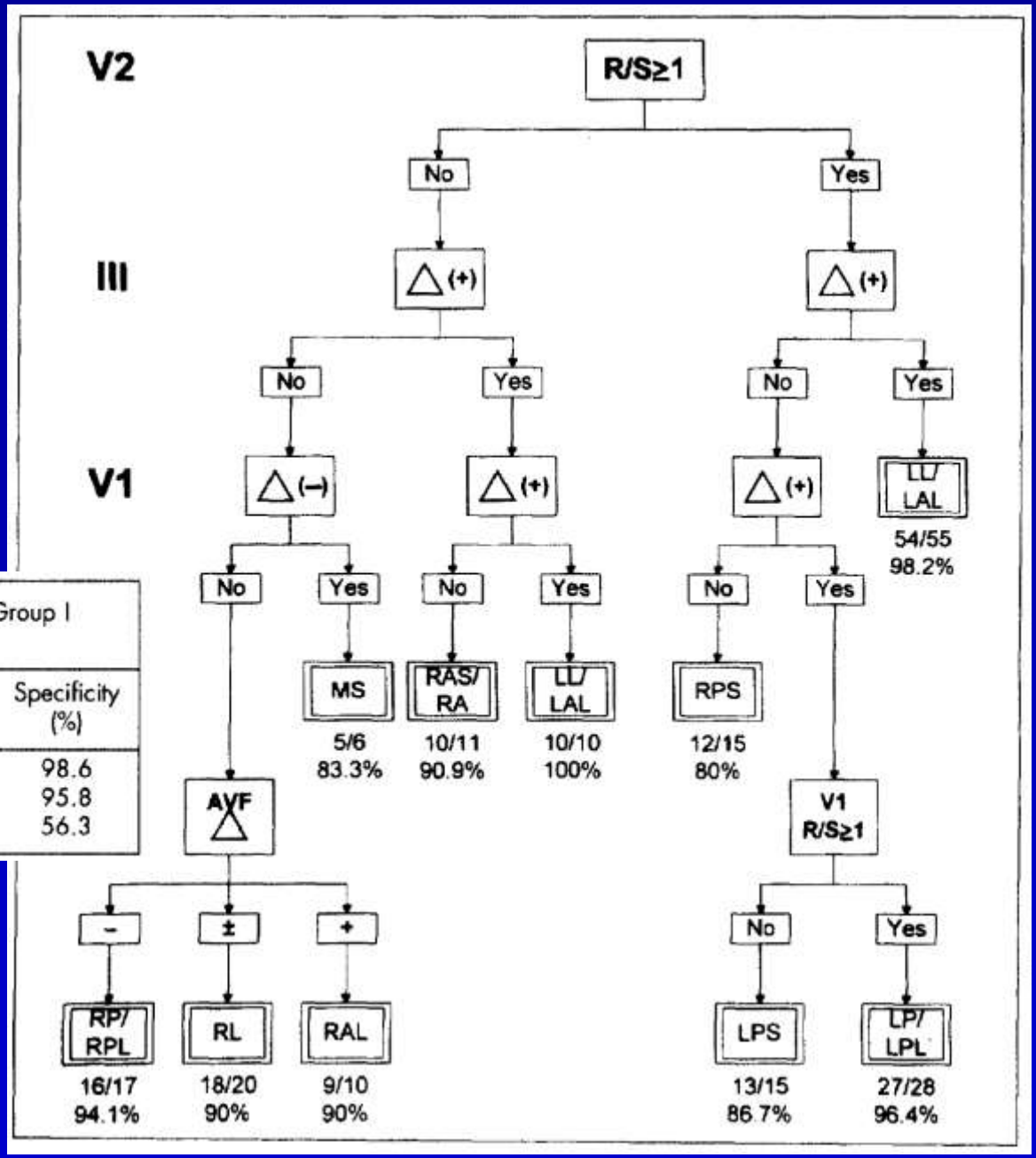
FIGURE 3. Delta wave axis in the frontal plane of the initial 182 patients. The axis of the delta waves for each region showed much overlap, and was not very helpful for differentiation. Left lateral (LL)/left anterolateral (LAL) (range +60° to +120°); left posterior (LP)/left posterolateral (LPL) (range −60° to +30°); left posteroseptal (LPS) (range −60° to +30°); midseptal (MS) (range −30° to +30°); right anterolateral (RAL) (range +15° to +45°); right anteroseptal (RAS)/right anterior (RA) (range +30° to +60°); right lateral (RL) (range −30° to +30°); right posterior (RP)/right posterolateral (RPL) (range −60° to −15°); right posteroseptal (RPS) (range −75° to +15°).



Chiang CE et al. Am J Cardiol 1995;76:40.

TABLE II Difference in Precordial R/S Ratio Between Group I and II Pathways

	Group I (n = 111)	Group II (n = 71)	Sensitivity (%)	Specificity (%)
V_1 R/S ≥1	68	1	61.3	98.6
V_2 R/S ≥1	105	3	94.6	95.8
V_3 R/S ≥1	111	31	100	56.3



Chiang CE et al. Am J Cardiol 1995;76:40.

Left Free Wall Pathway

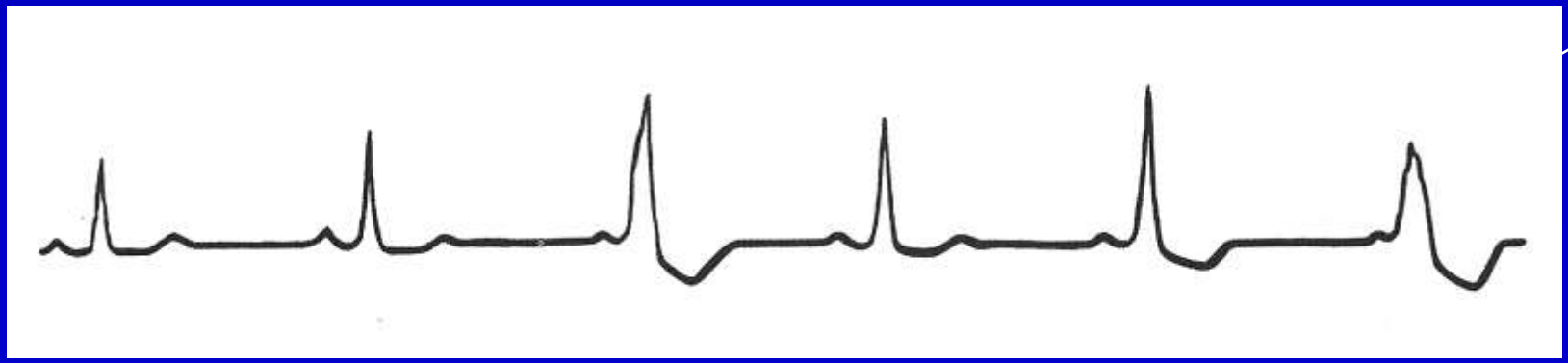
- Most common
- Negative delta waves in I and L and positive delta in inferior leads and all precordial leads (Josephson p. 356)
- Negative delta waves in I, aVL, or V6 and a “pseudo-right bundle branch block” QRS complex appearance with positive QRS complex (Rs wave) in V1 (Prystowsky in Zipes p. 873)
- Step 1: If the delta wave in lead I is (-) or (+-) or the R/S in lead V1 is >1, a left free-wall AP is present (Surawicz p. 479 after Arruda)

Wolff-Parkinson-White Syndrome (WPW)

- Short PR interval (<0.12 sec) in 75-90%
- Wide QRS complex (≥ 0.11 sec) in 65-75%
- Slurred initial forces of QRS - Delta wave
- Secondary ST segment and T wave abnormality (discordant to Delta wave)
- Frequent association of paroxysmal tachycardia, usually supraventricular

Wolff-Parkinson-White Syndrome (WPW) - 2

- Every beat is a fusion beat
  - Part of QRS from AV node and normal His-Purkinje system
  - Part of QRS from conduction through the accessory AV connection (“Bundle of Kent”) from atrial muscle to ventricular muscle
  - Variable conduction depends on how much of the ventricle is excited from the normal versus the



Wolff-Parkinson-White Syndrome (WPW) - 3

- Tachycardia is often due to electrical activity travelling in a circular pathway
- One Possible Pathway
  - AV node
  - Atrial muscle
  - Accessory pathway
  - Ventricular muscle
- "Circus movement"
- Atrioventricular reentrant tachycardia (AVRT)

Tachycardias in WPW Syndrome

- Accessory pathway integral to circuit
  - Orthodromic AVRT (most common)
    - With or without functional bundle branch block (ipsilateral, slows rate)
  - Pre-excited reciprocating tachycardias
  - Antidromic AV reentrant tachycardias
  - AVRT with multiple pathways
- Accessory pathway passive, not essential
  - AVNRT
  - AVRT with second bystander accessory pathway
  - Aflutter or Fibrillation
  - VT

Zipes and Jalife, 4th ed, 2004, p. 869-878

Tachycardias in WPW Syndrome

- Accessory pathway integral to circuit
  - Orthodromic AVRT (most common)
    - With or without functional bundle branch block (ipsilateral, slows rate)
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  - Antidromic AV reentrant tachycardias
  - AVRT with multiple pathways
- Accessory pathway passive, not essential
  - AVNRT
  - AVRT with second bystander accessory pathway
  - Atrial flutter or fibrillation
  - VT

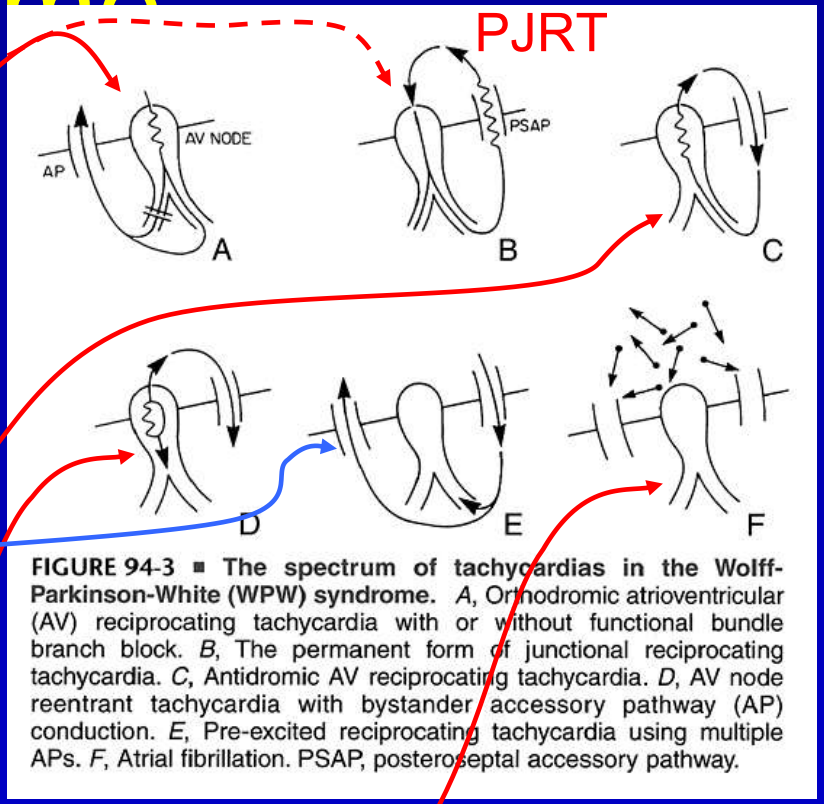


FIGURE 94-3 ■ The spectrum of tachycardias in the Wolff-Parkinson-White (WPW) syndrome. *A*, Orthodromic atrioventricular (AV) reciprocating tachycardia with or without functional bundle branch block. *B*, The permanent form of junctional reciprocating tachycardia. *C*, Antidromic AV reciprocating tachycardia. *D*, AV node reentrant tachycardia with bystander accessory pathway (AP) conduction. *E*, Pre-excited reciprocating tachycardia using multiple APs. *F*, Atrial fibrillation. PSAP, posteroseptal accessory pathway.

Zipes and Jalife, 4th ed, 2004, p. 869-878

Orthodromic AVRT and BBB

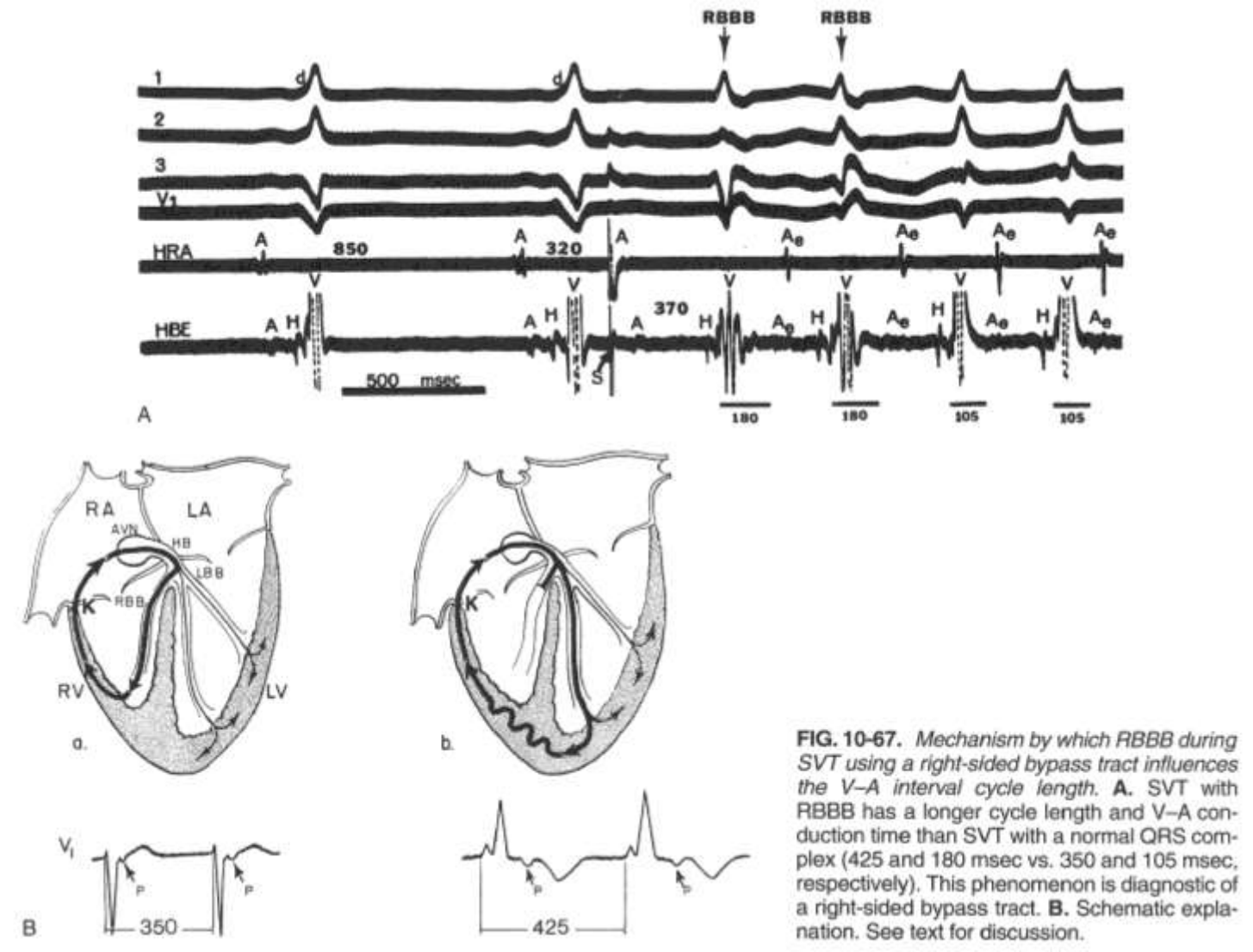


FIG. 10-67. *Mechanism by which RBBB during SVT using a right-sided bypass tract influences the V–A interval cycle length.* **A.** SVT with RBBB has a longer cycle length and V–A conduction time than SVT with a normal QRS complex (425 and 180 msec vs. 350 and 105 msec, respectively). This phenomenon is diagnostic of a right-sided bypass tract. **B.** Schematic explanation. See text for discussion.

Josephson ME. Clinical Cardiac Electrophysiology 2002; p. 370.

PJRT, or PAVRT

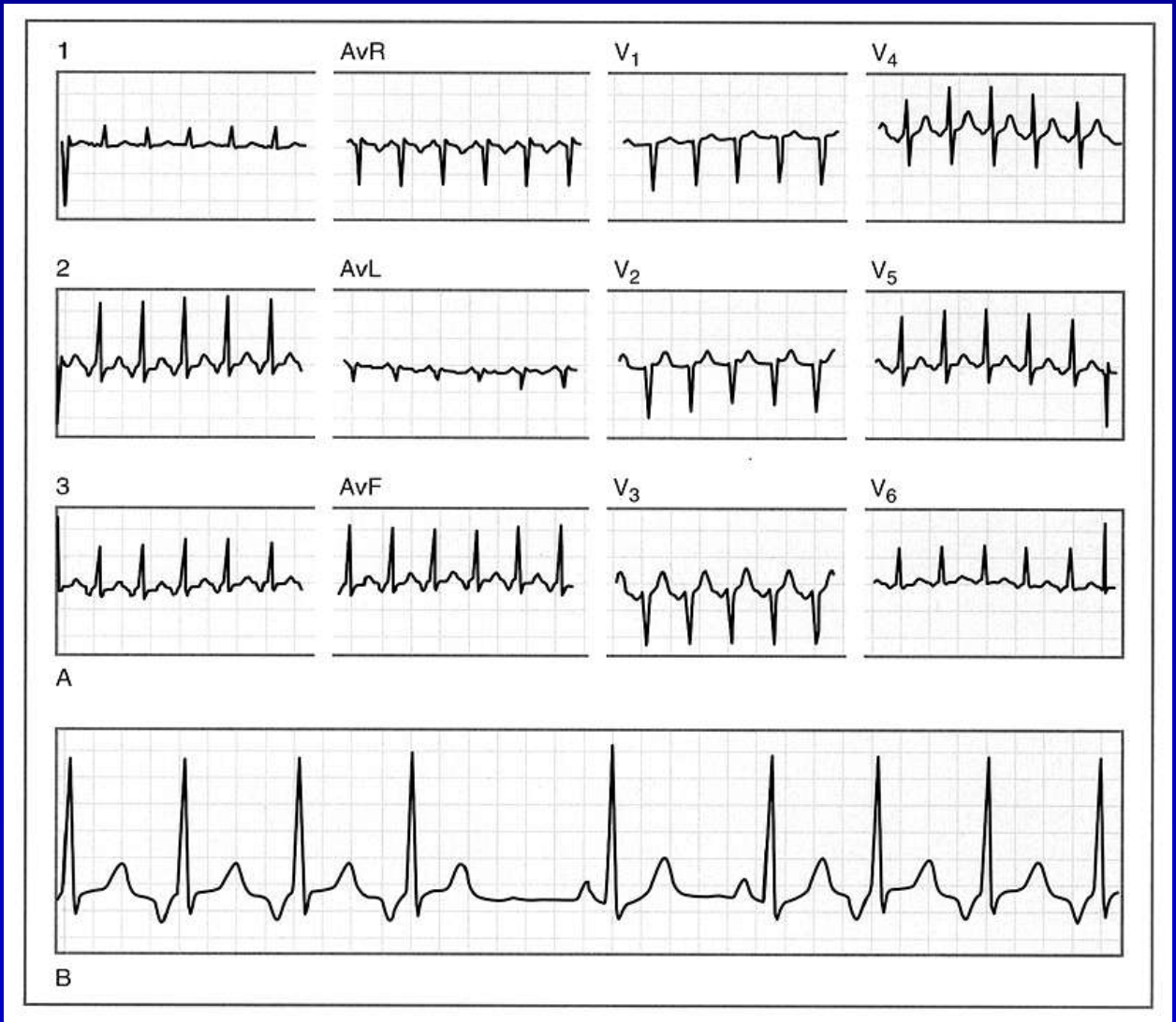
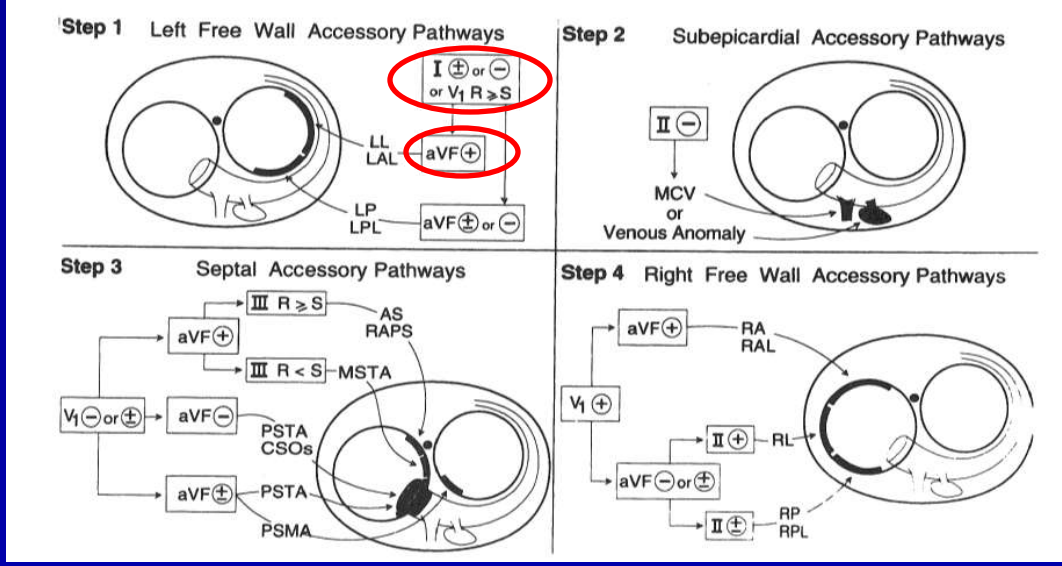
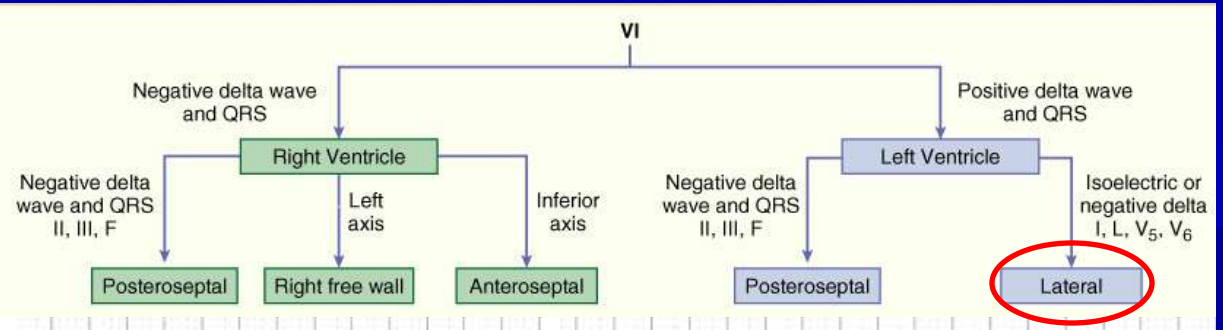


FIGURE 94-5 ■ Surface electrocardiogram of a patient with the permanent form of junctional reciprocating tachycardia. *A*, The 12-lead electrocardiogram illustrates the essential features of the tachycardia with typical negative P waves in leads II, III, and aVF and an R-P interval longer than the P-R interval. *B*, The tachycardia was transiently terminated by right carotid sinus massage. Termination without a retrograde P wave indicated block occurred in the retrograde limb and illustrated the atrioventricular node–like behavior of the accessory pathway. With acceleration of the sinus rate, there was spontaneous resumption of tachycardia.

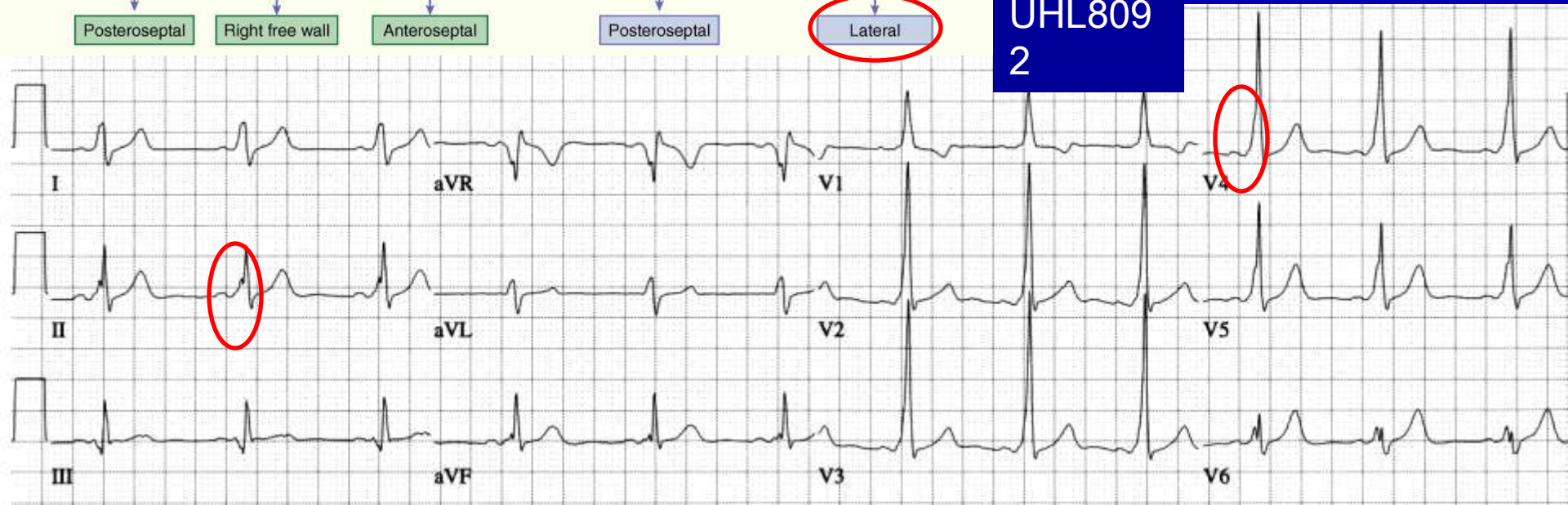
Zipes and Jalife, 4th ed, 2004, p. 869-878

- Left lateral or anterolateral pathway

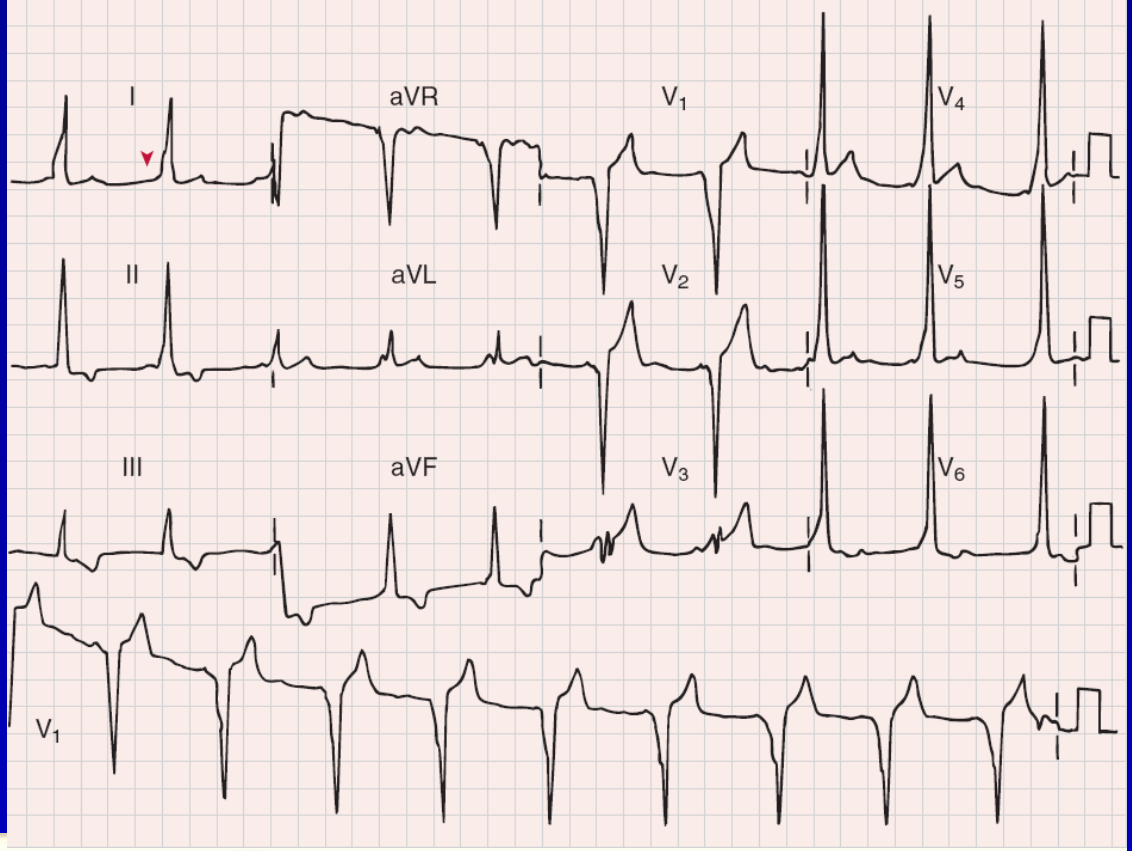


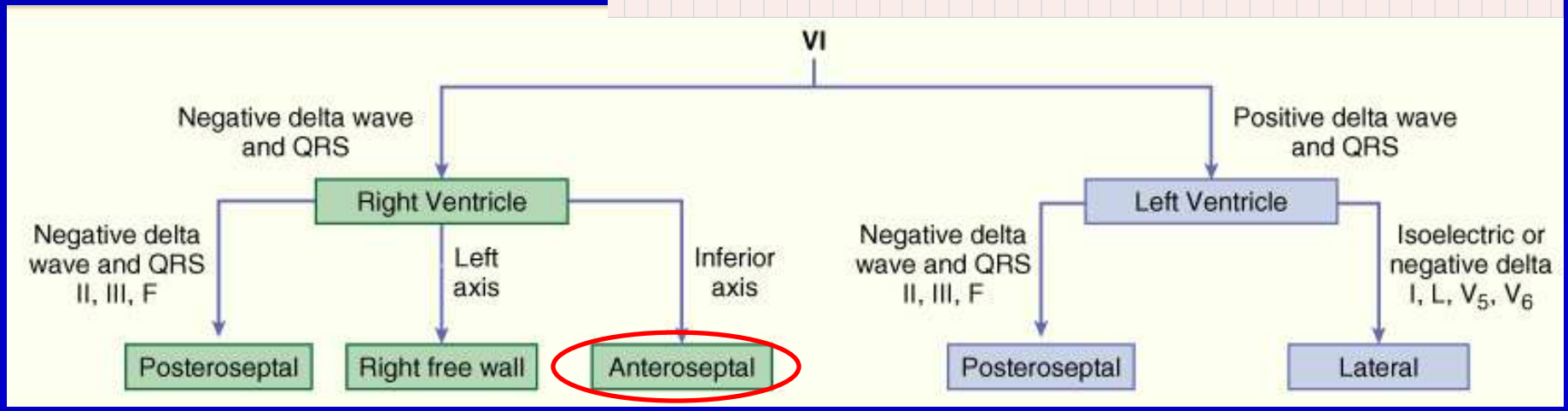


UHL809 2



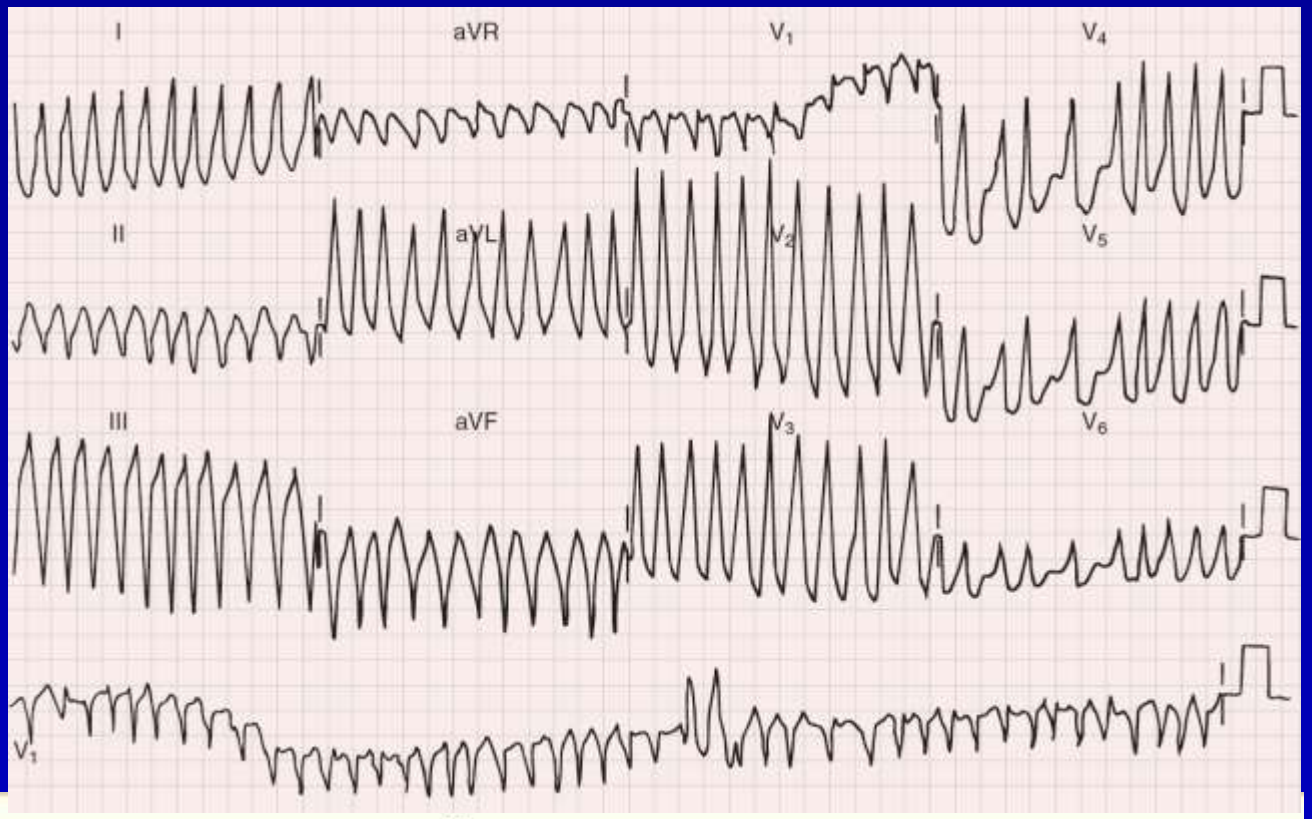
Right anteroseptal accessory pathway, characteristic inferior axis, delta wave is negative in V1 and V2, upright in I, II, aVL, and aVF, isoelectric in III, negative in aVR

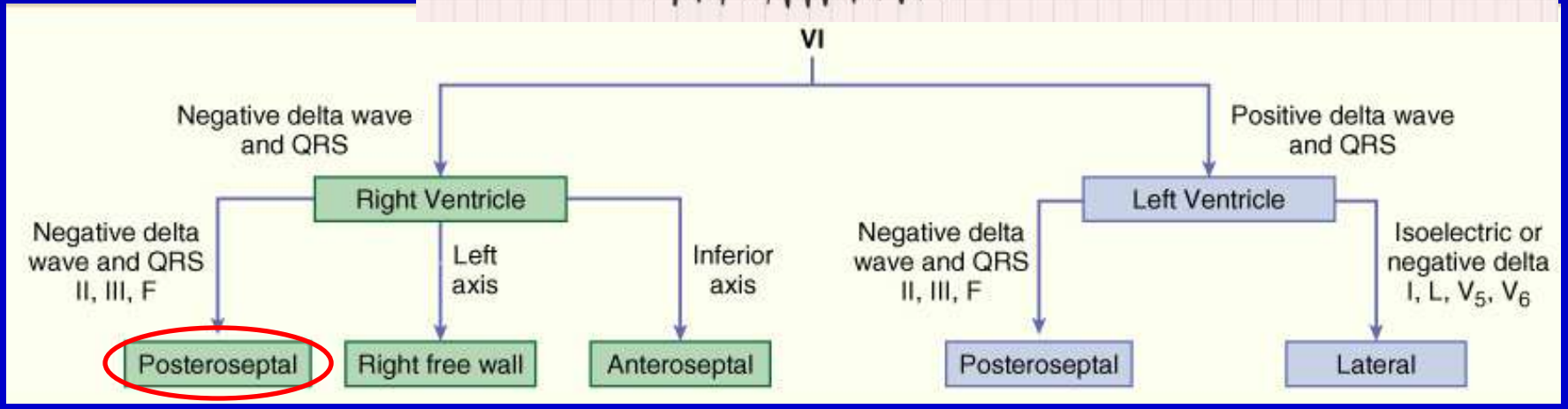




Braunwald, Ch. 32, “Specific Arrhythmias: Diagnosis and Treatment”, Olgin JE and Zipes DP. p. 830, 2005

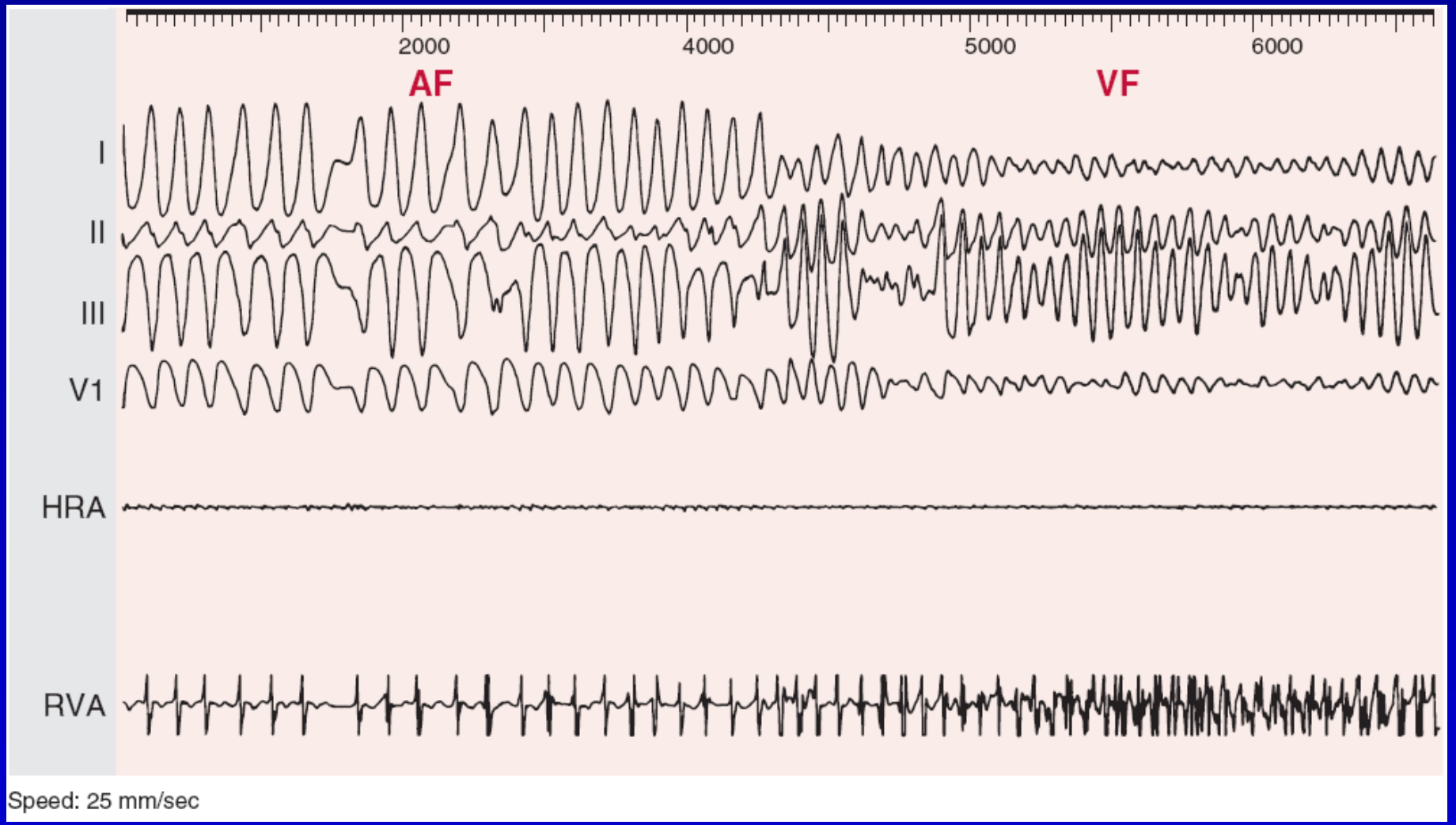
Right posteroseptal accessory pathway. negative delta in II, III, and aVF, upright in I and aVL localize the pathway to posteroseptal, and negative delta in V1 and rapid transition in V2 pinpoints to right posteroseptal; AFib





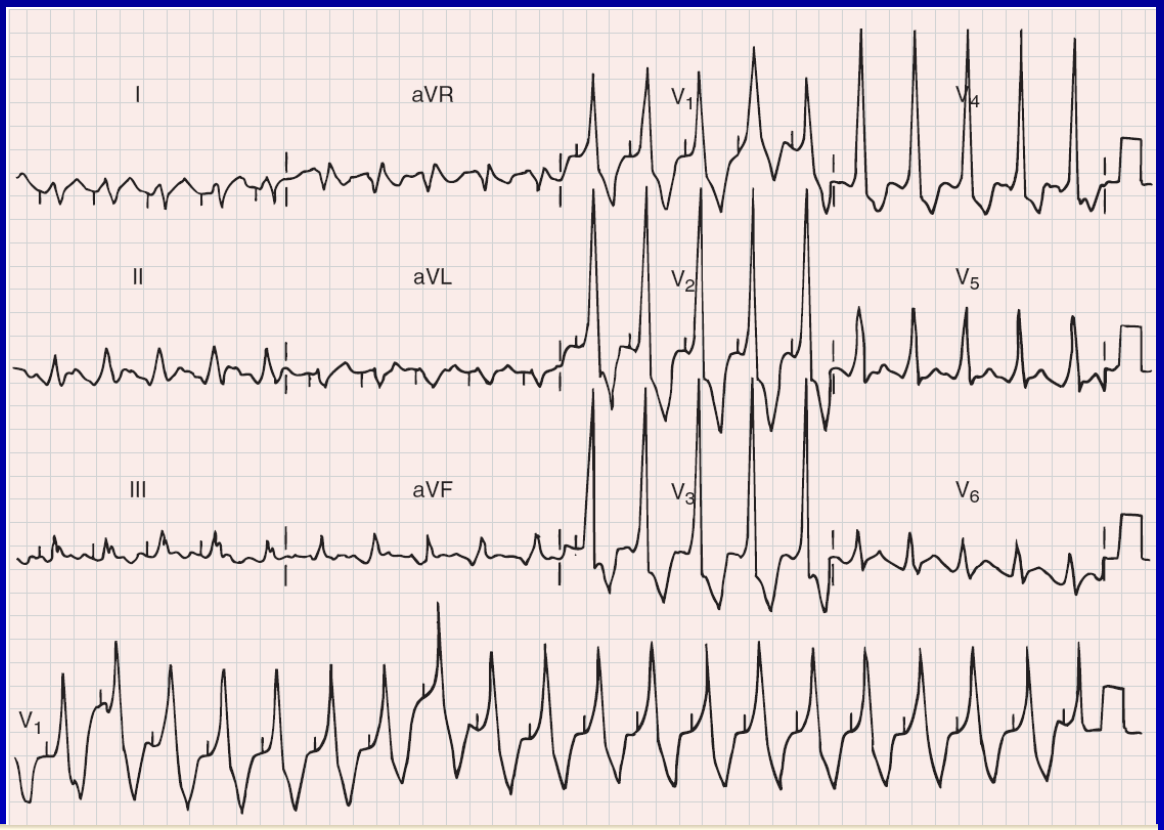
Braunwald, Ch. 32, “Specific Arrhythmias: Diagnosis and Treatment”, Olgin JE and Zipes DP. p. 830, 2005

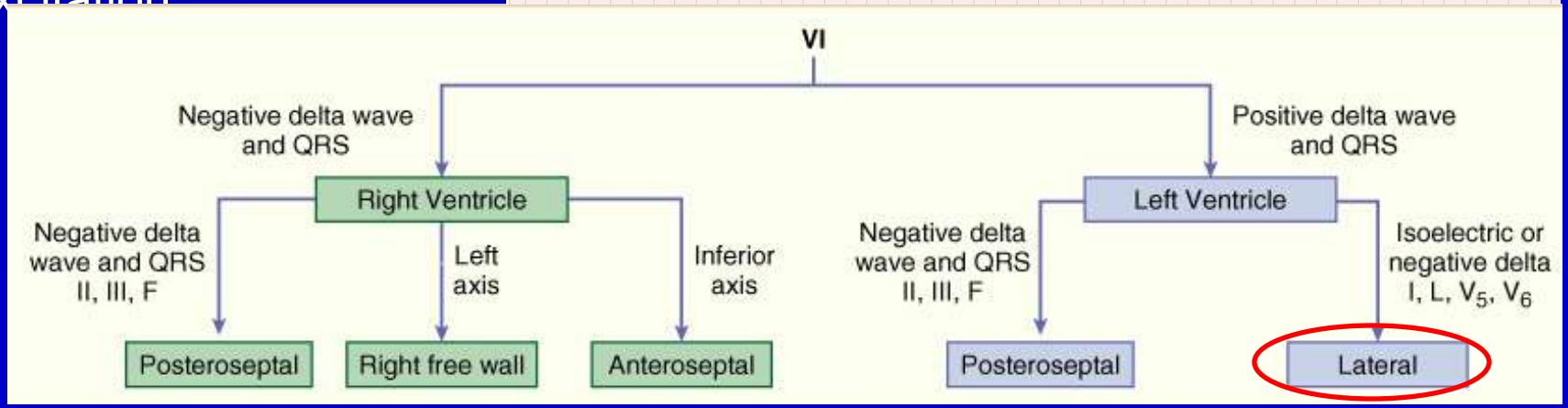
AF and WPW becoming VF



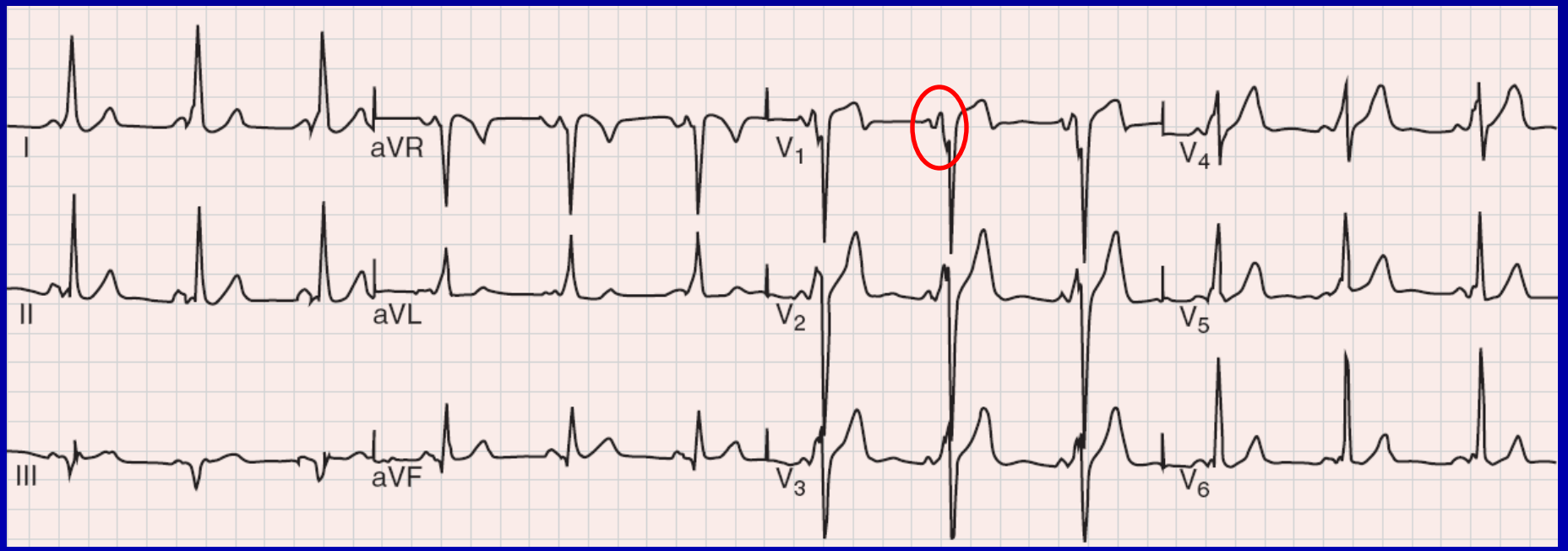
Braunwald, Ch. 32, “Specific Arrhythmias: Diagnosis and Treatment”, Olgin JE and Zipes DP. p. 836, 2005

Left lateral accessory pathway – positive delta in anterior precordial leads and in II, III, and aVF, positive or isoelectric in leads I and aVL, and isoelectric or negative in V5 and V6 is typical of a left lateral accessory pathway. Coronary sinus pacing was used to enhance preexcitation

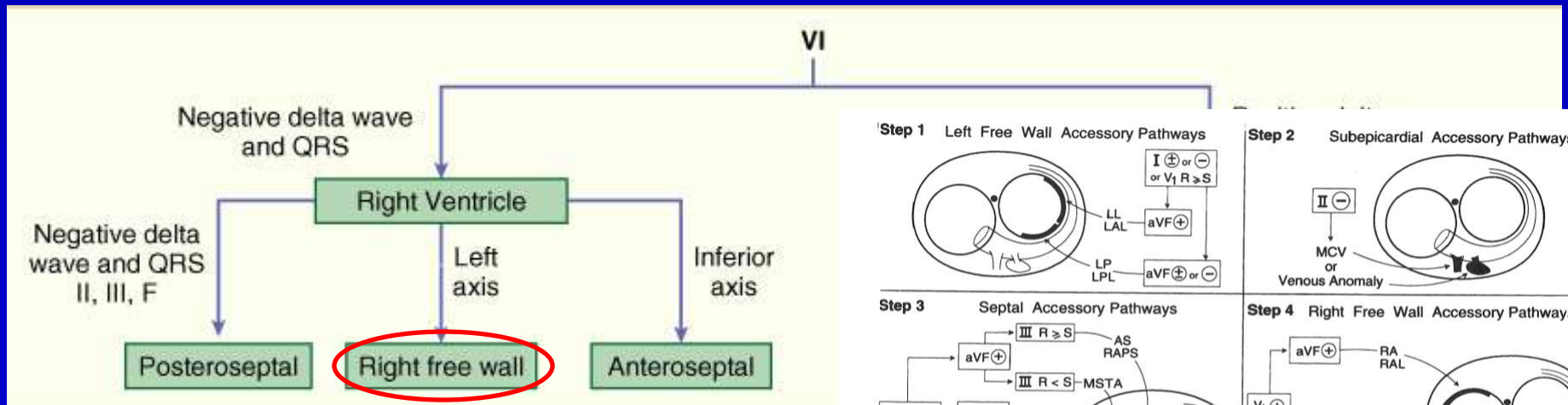




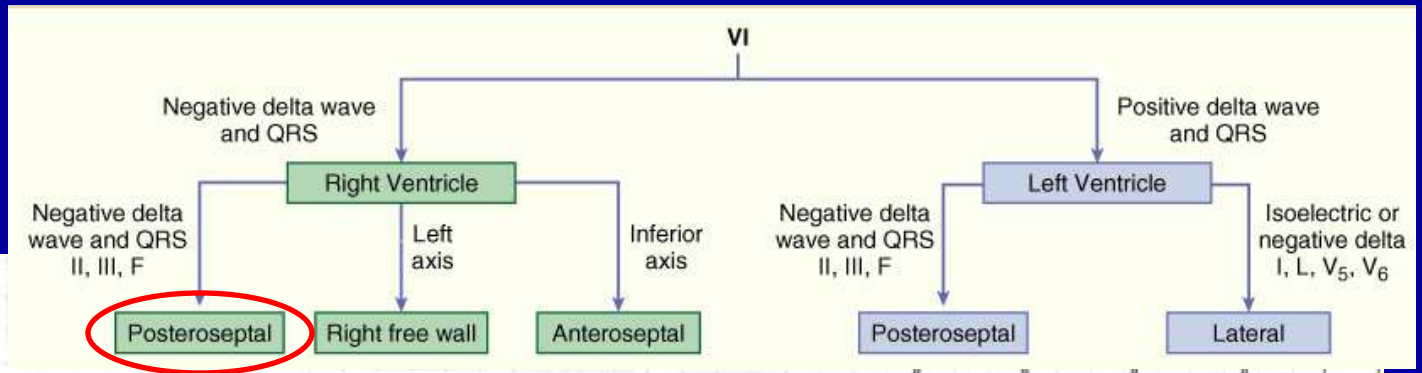
Braunwald, Ch. 32, “Specific Arrhythmias: Diagnosis and Treatment”, Olgin JE and Zipes DP. p. 830, 2005



Right free wall accessory pathway – predominantly negative delta in V1 and axis more leftward

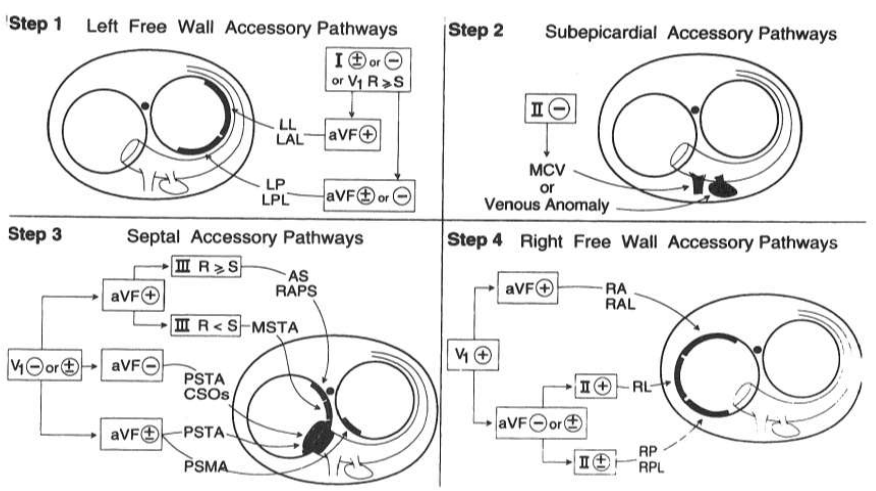


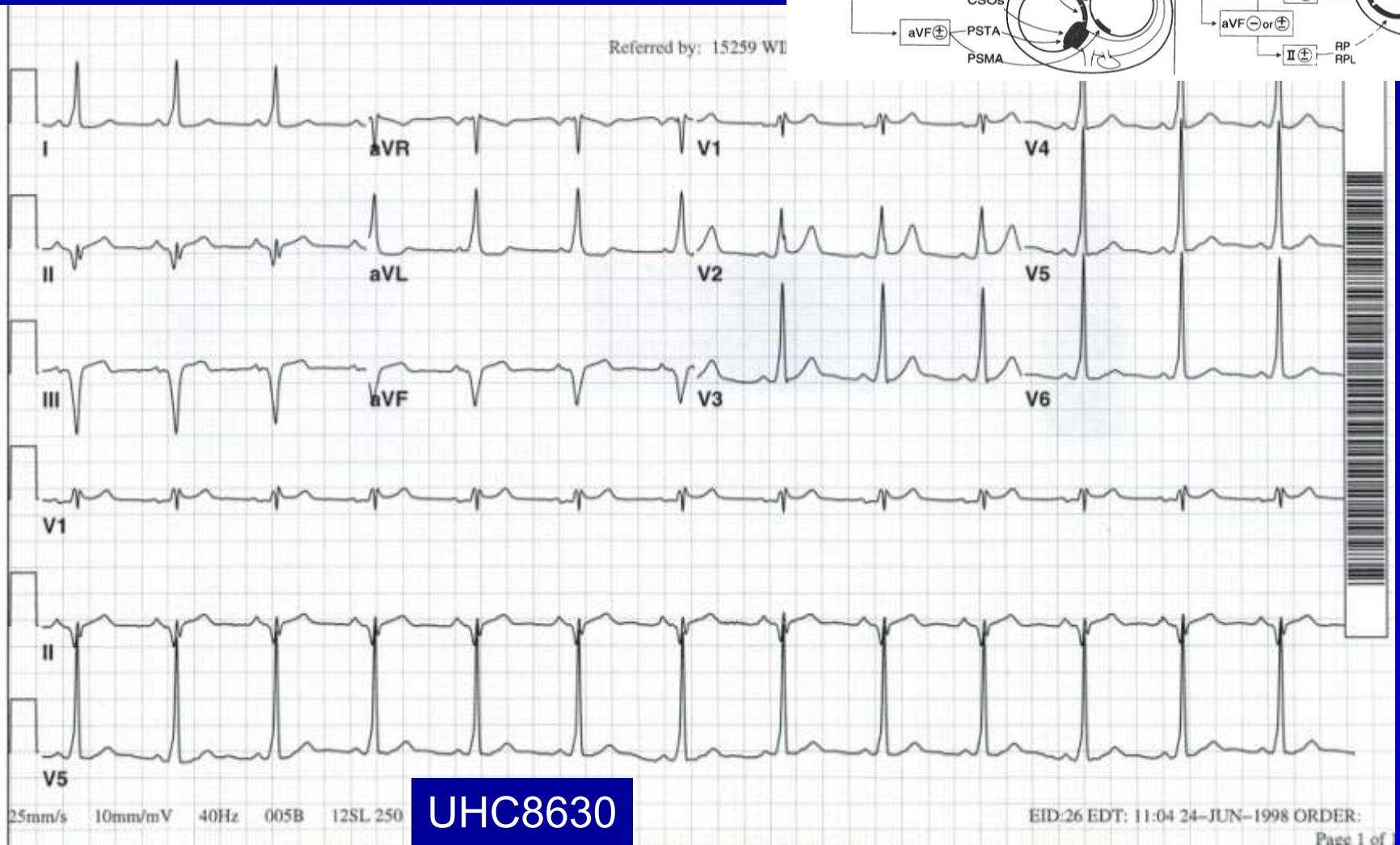
Braunwald, Ch. 32, "Specific Arrhythmias: and Zipes DP. p. 830, 2005

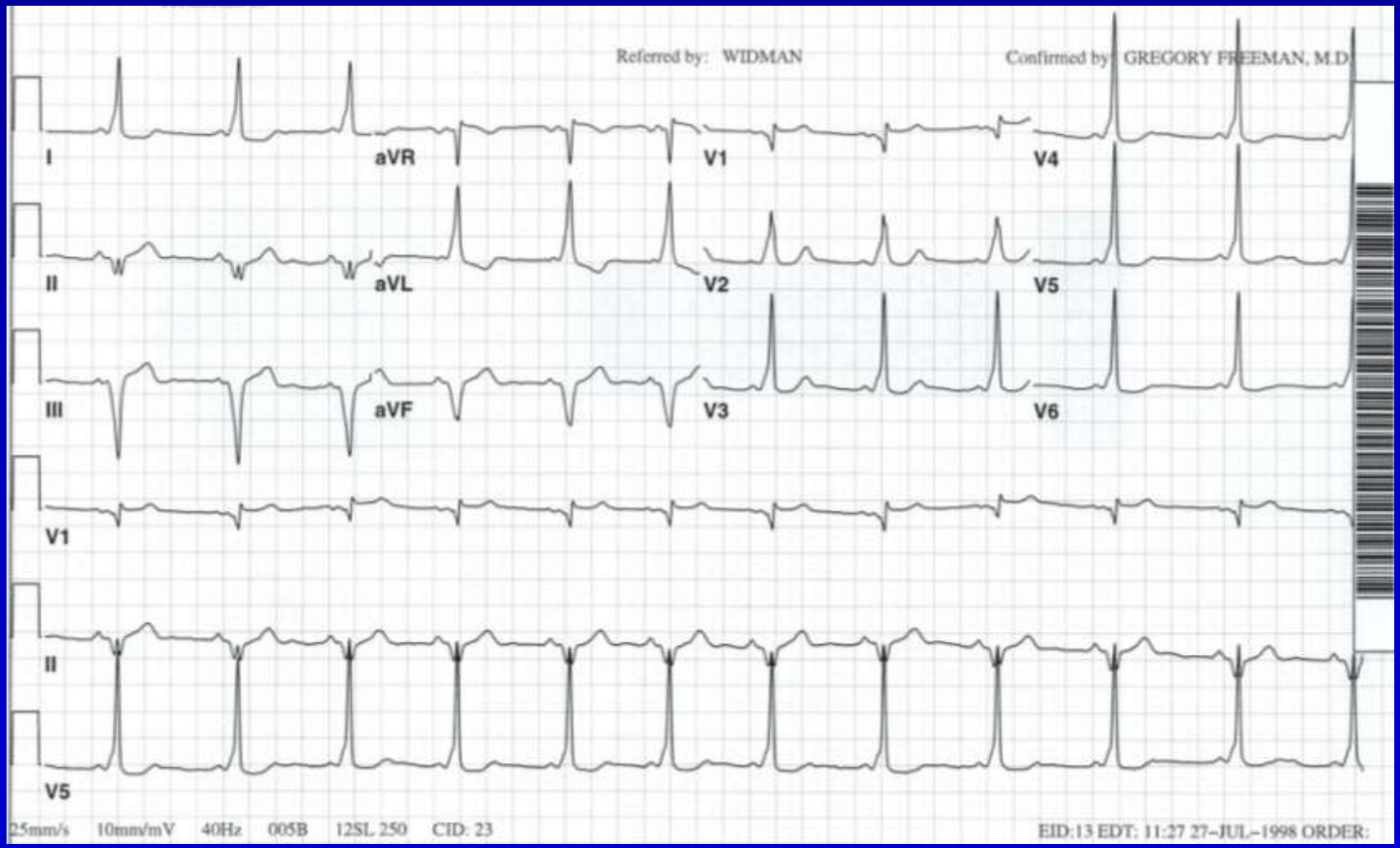


UHC3989, less pre-excited

Right posteroseptal – sudden transition from V1-V2 and negative in II, III, and aVF



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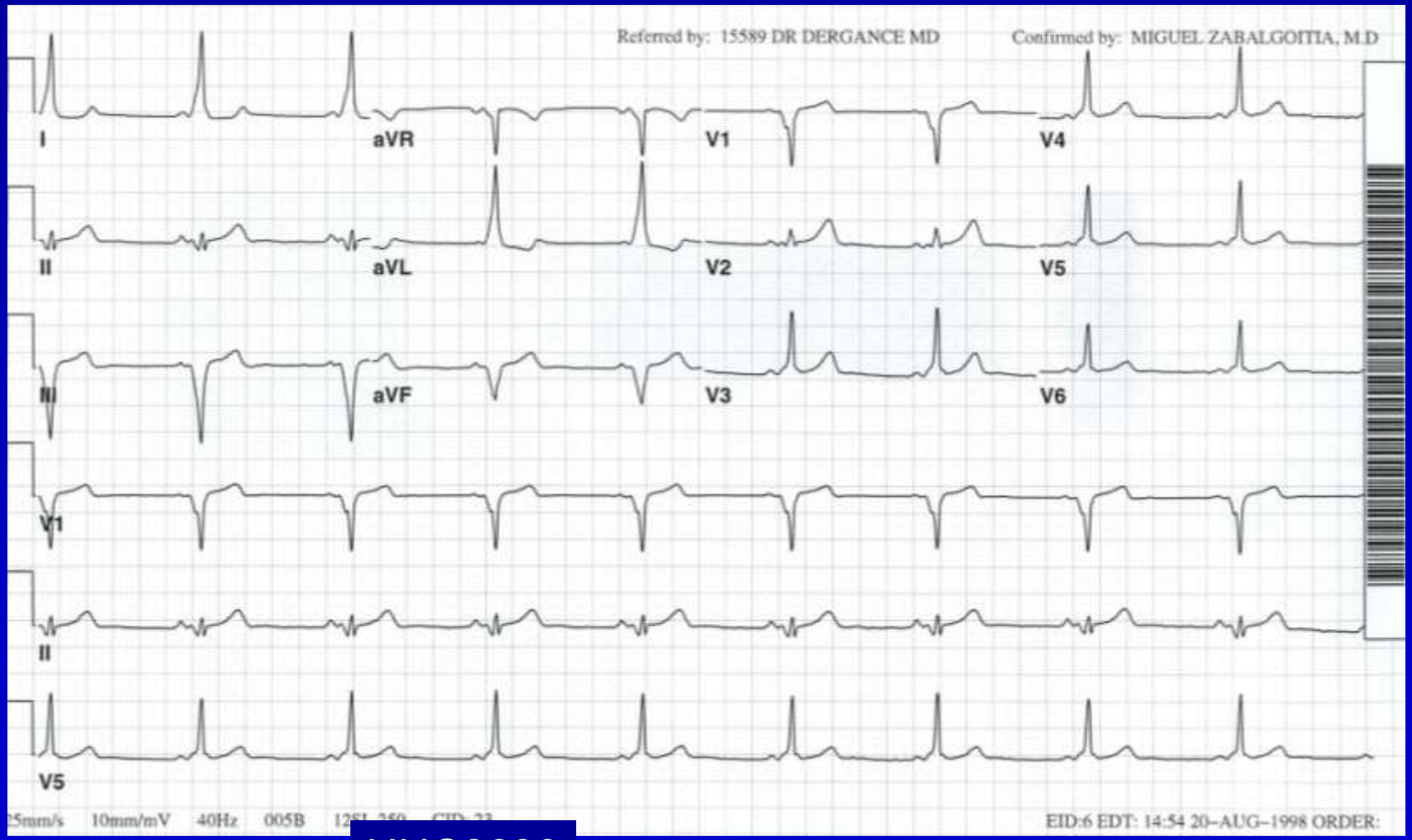
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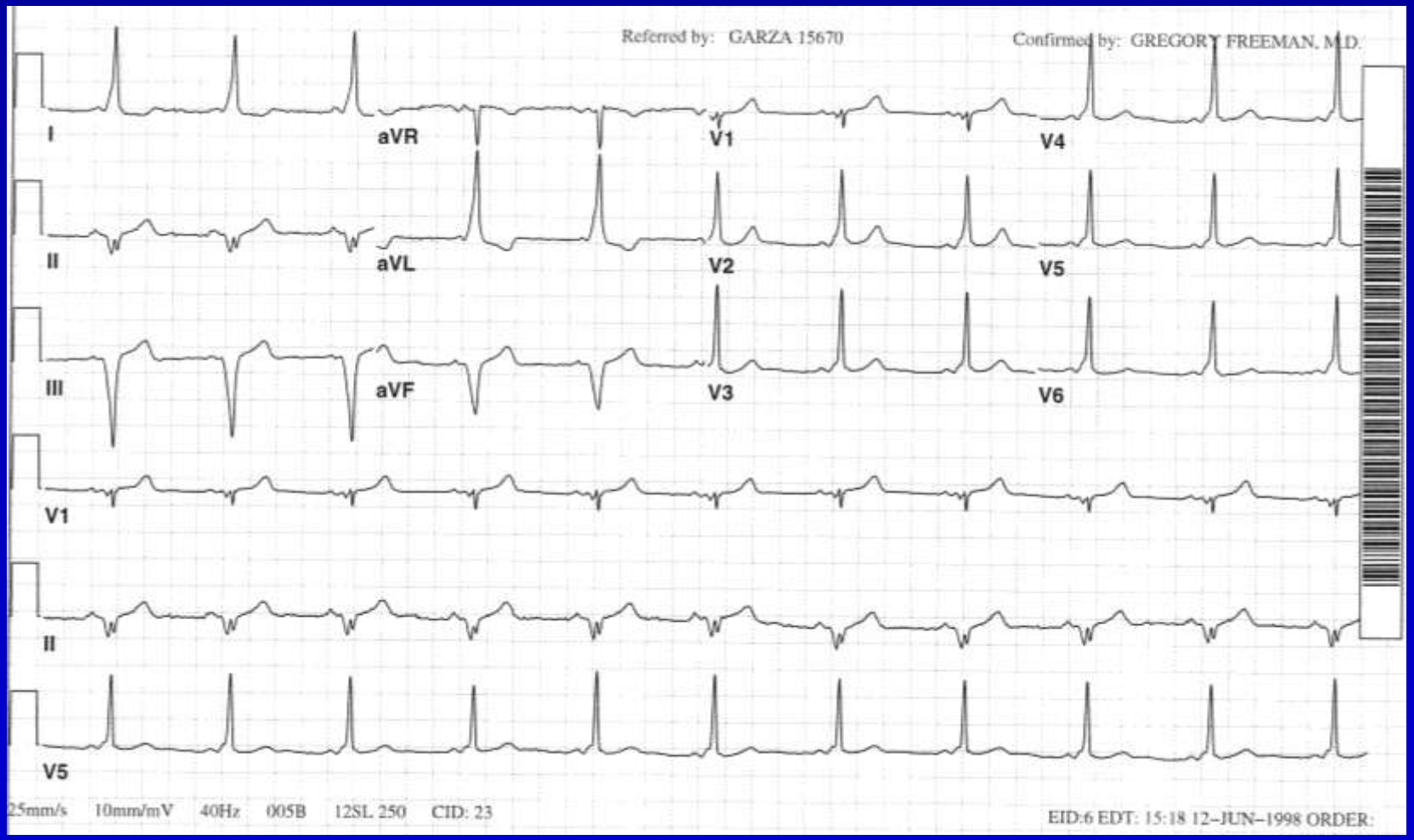
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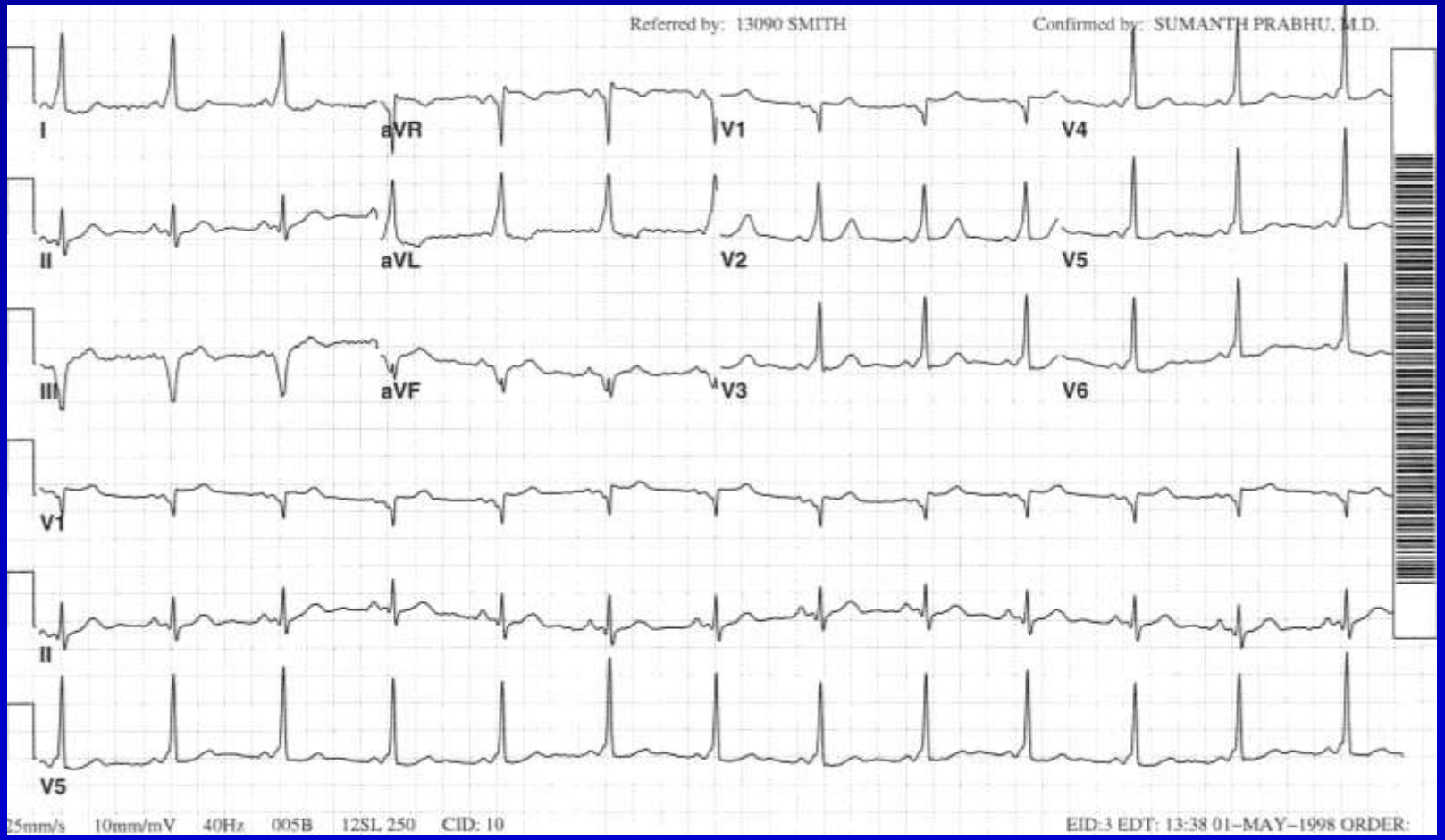
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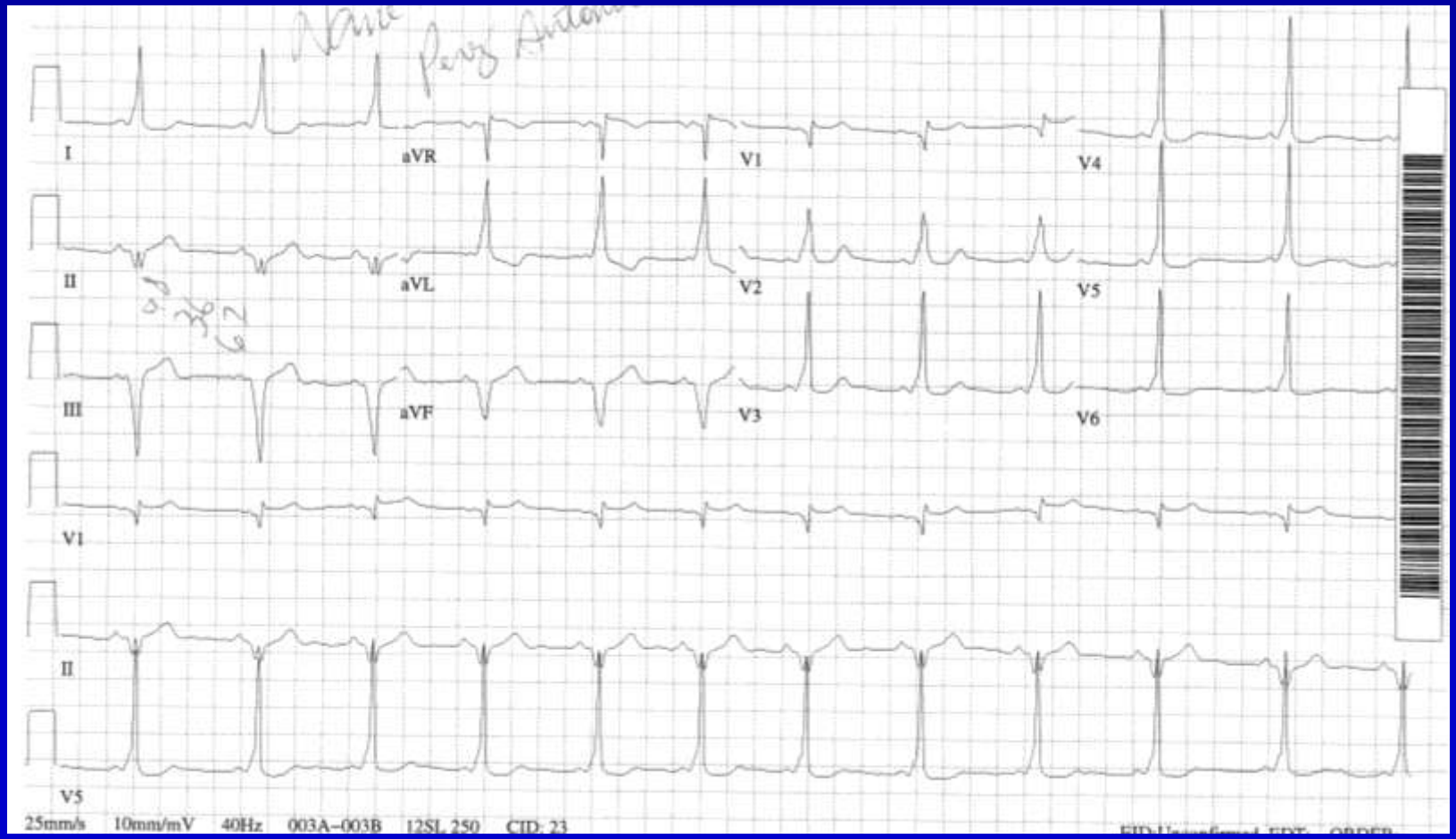
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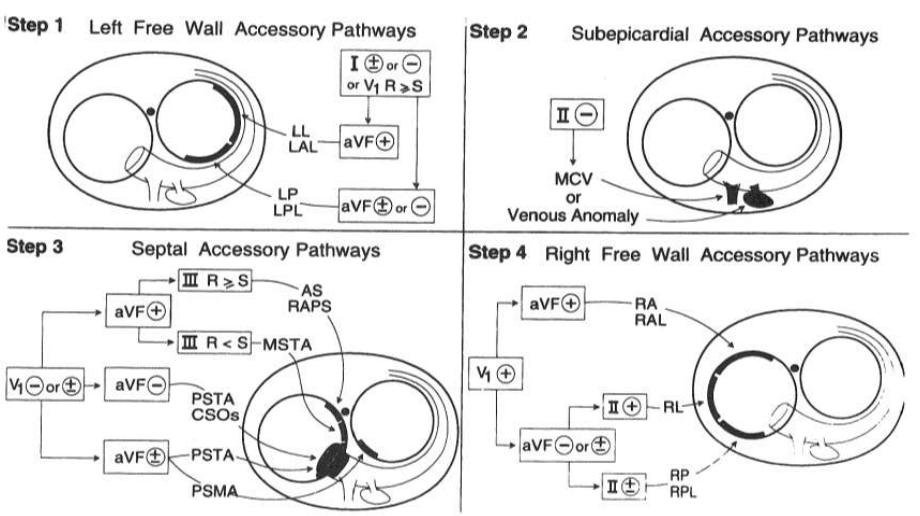
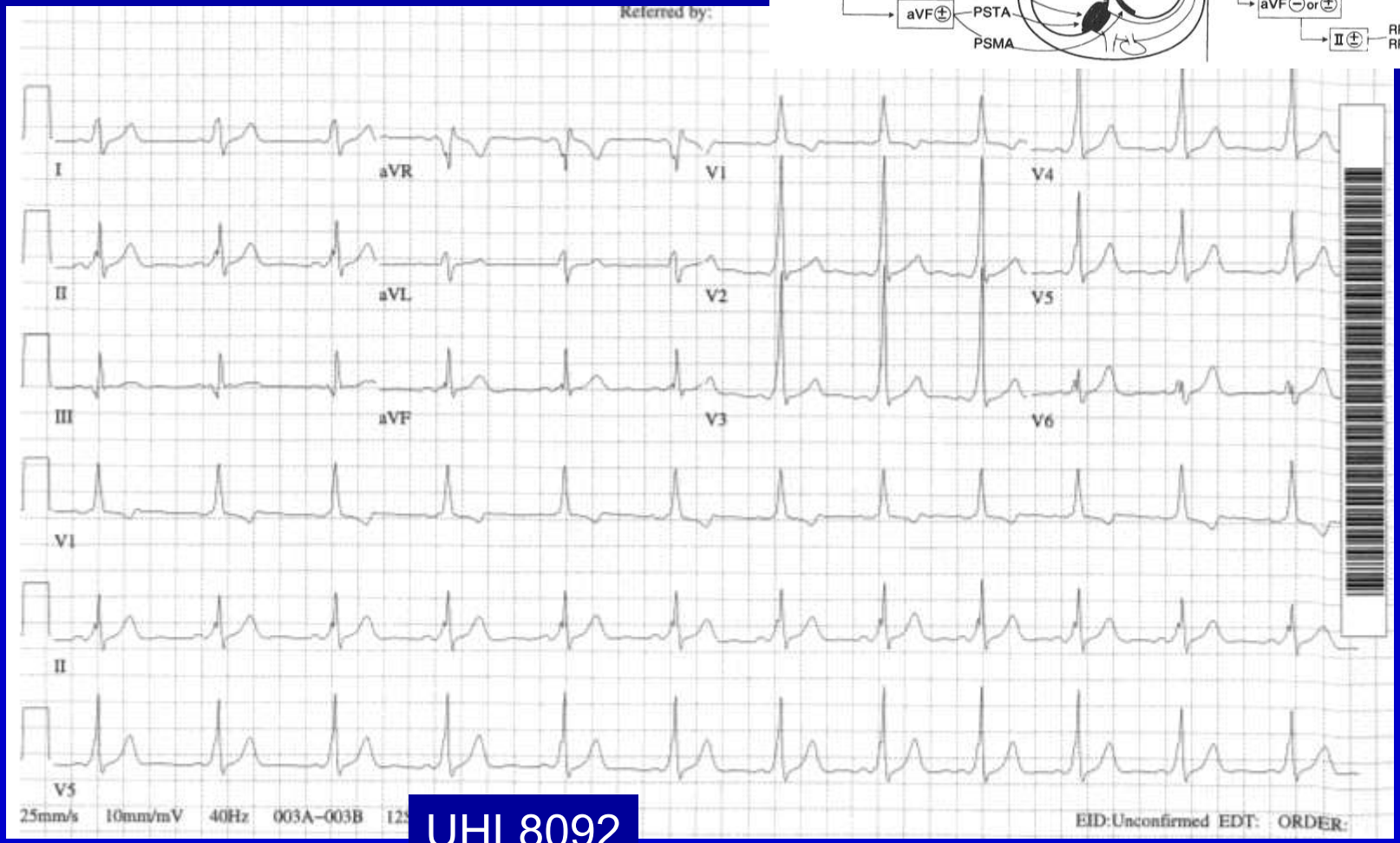
UHC8630

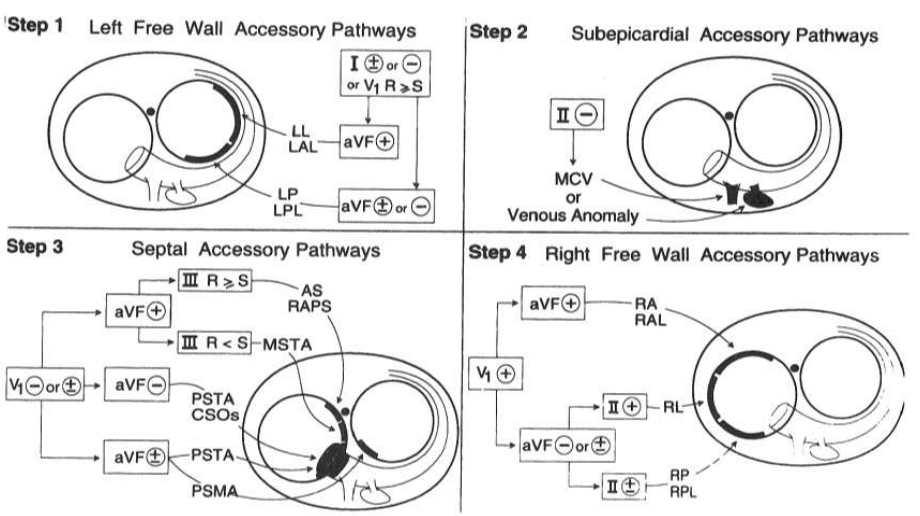
Left lateral

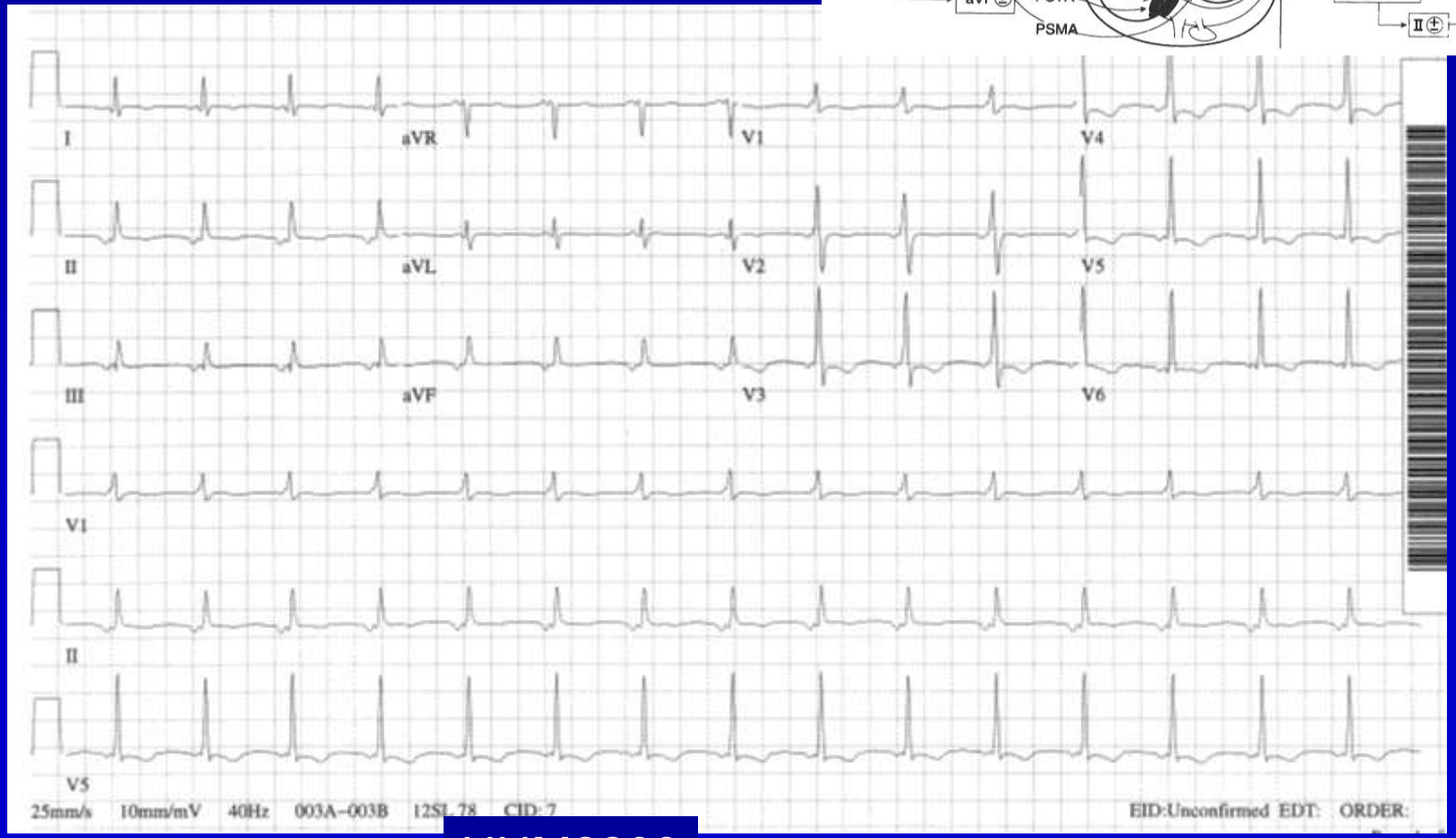




UHL8092

Read as WPW but questionable … short PR is because rhythm is not sinus





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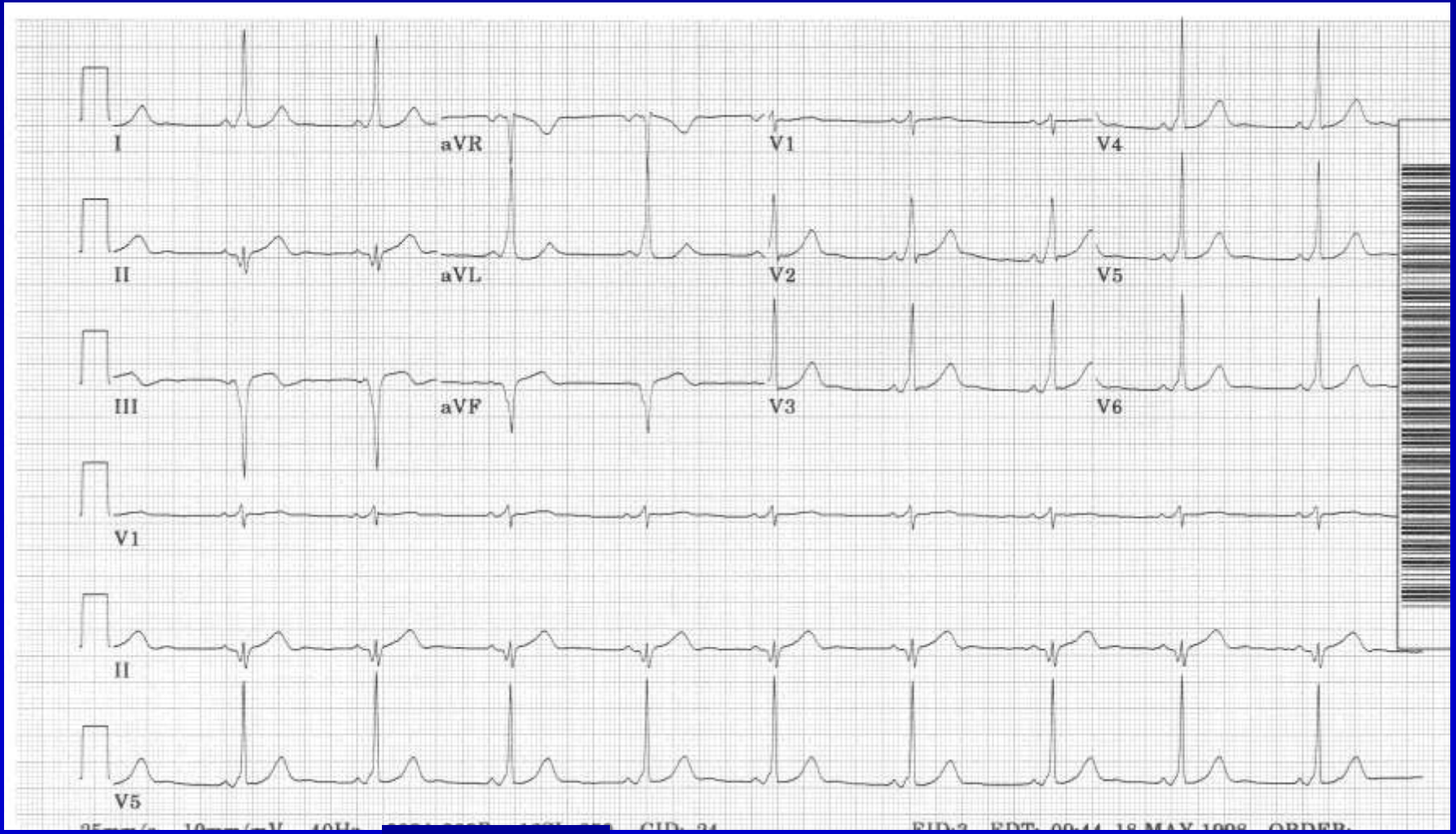

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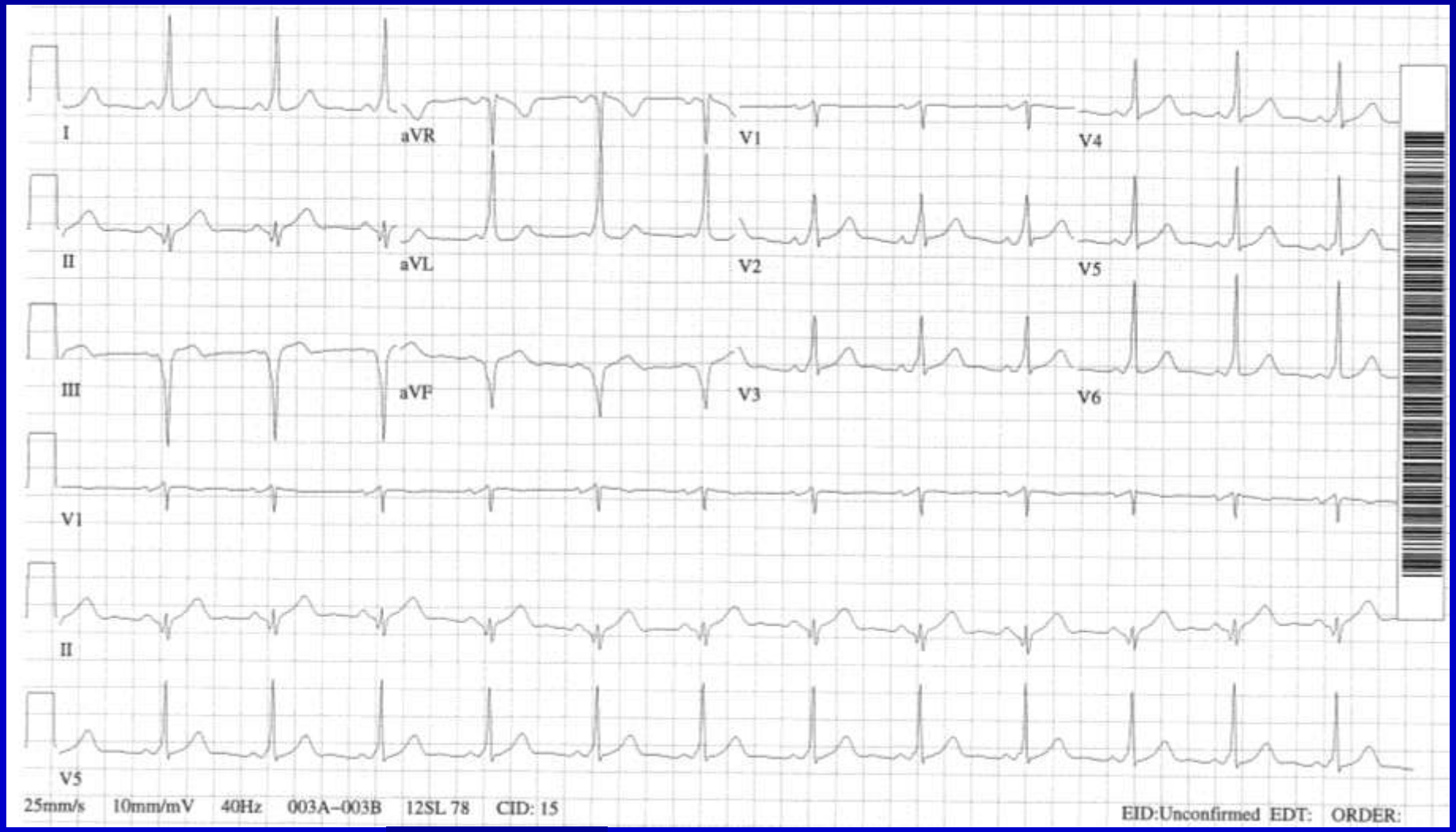
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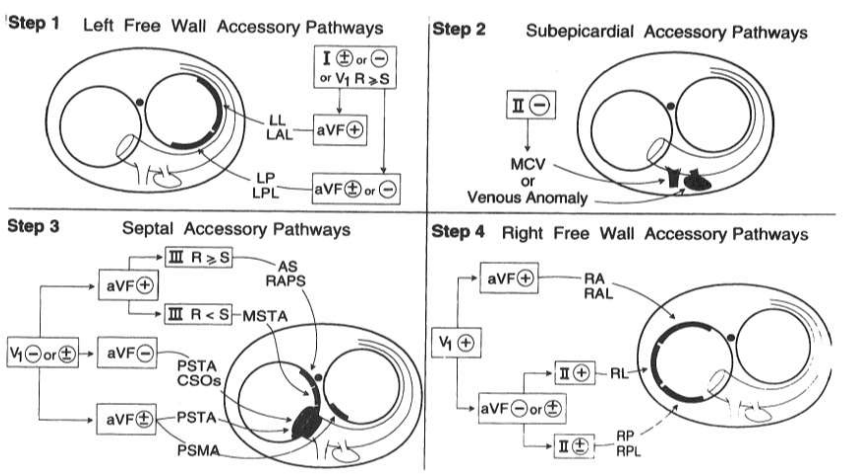
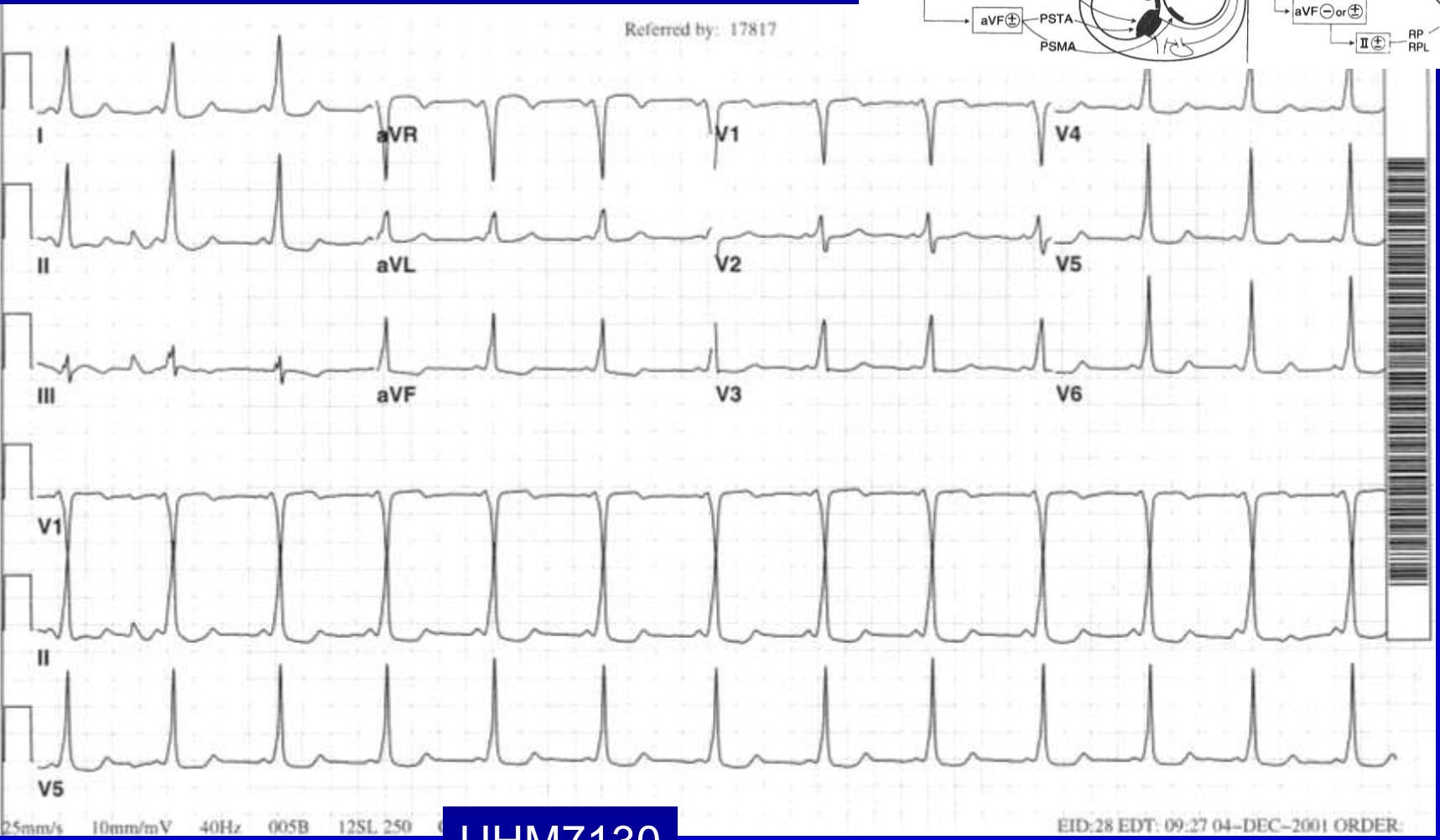
UHM3356

Right anterior paraseptal





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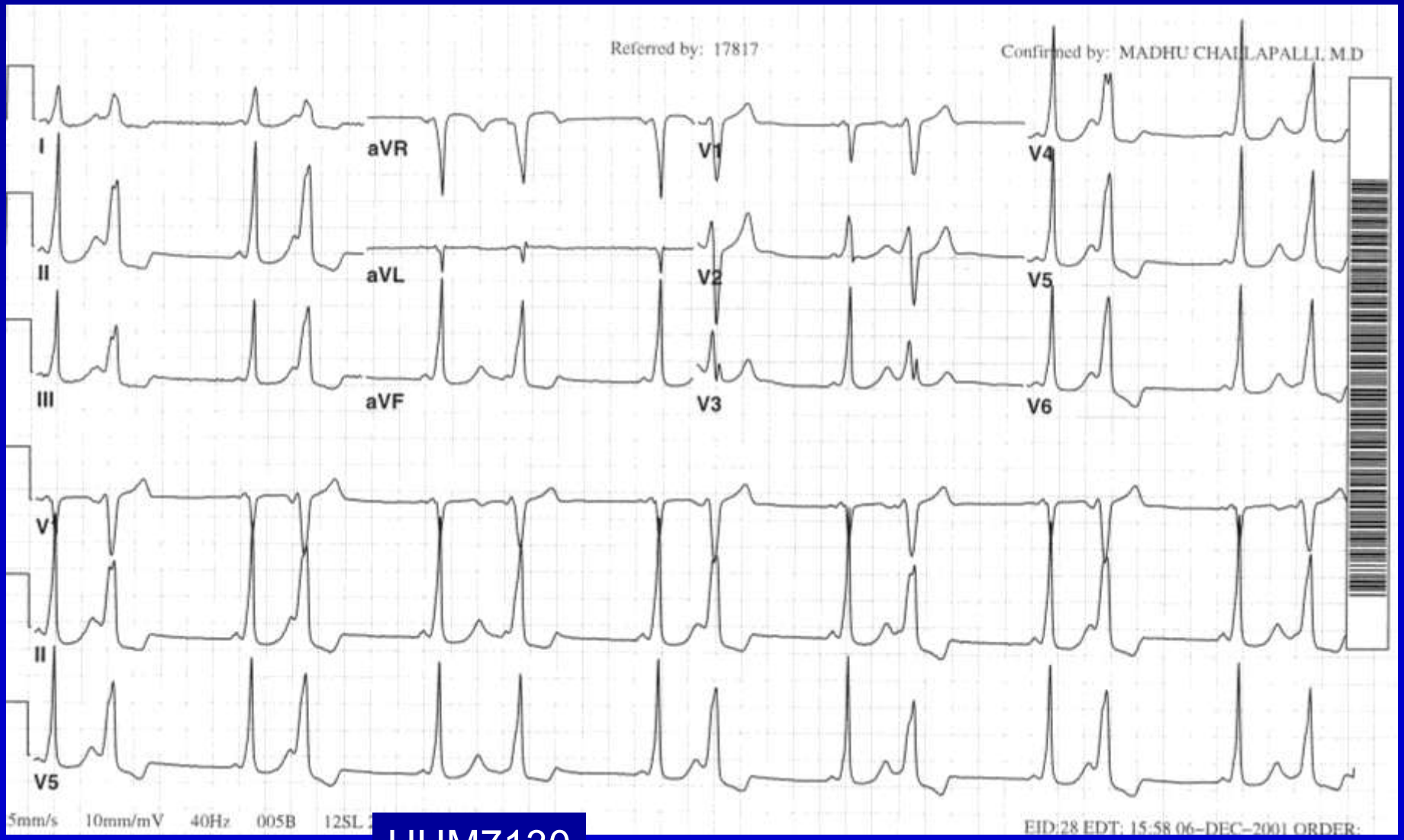
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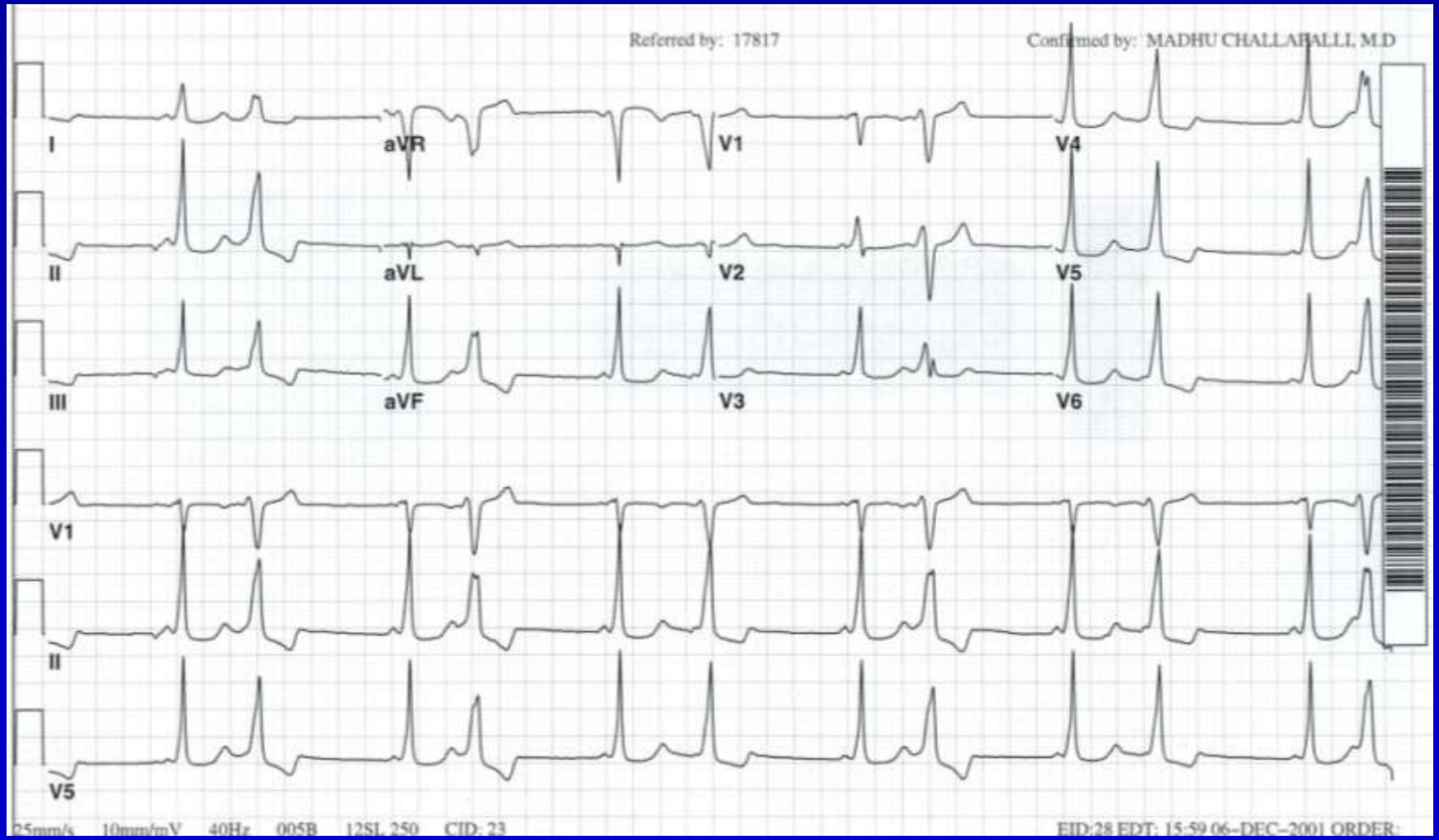
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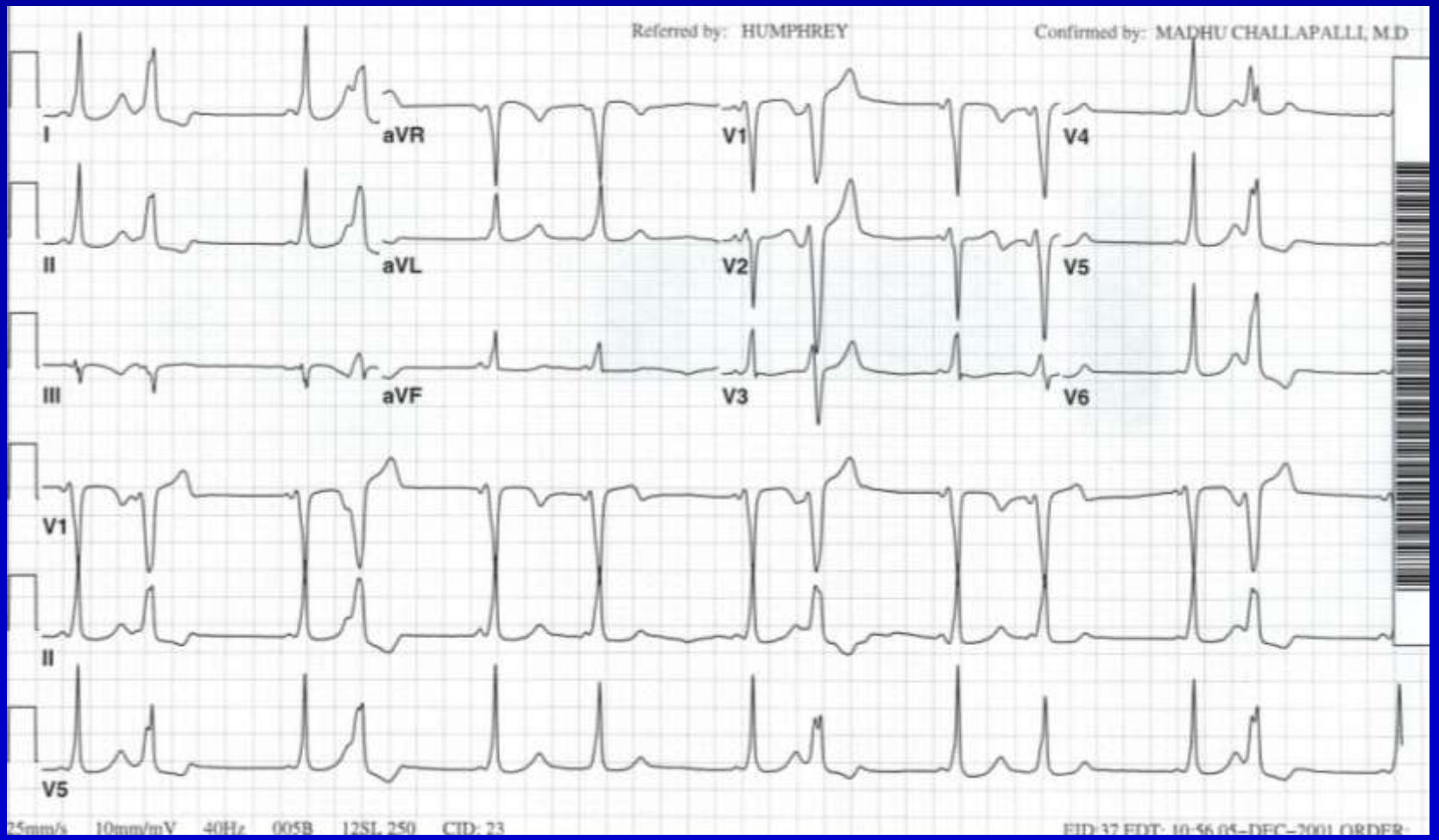
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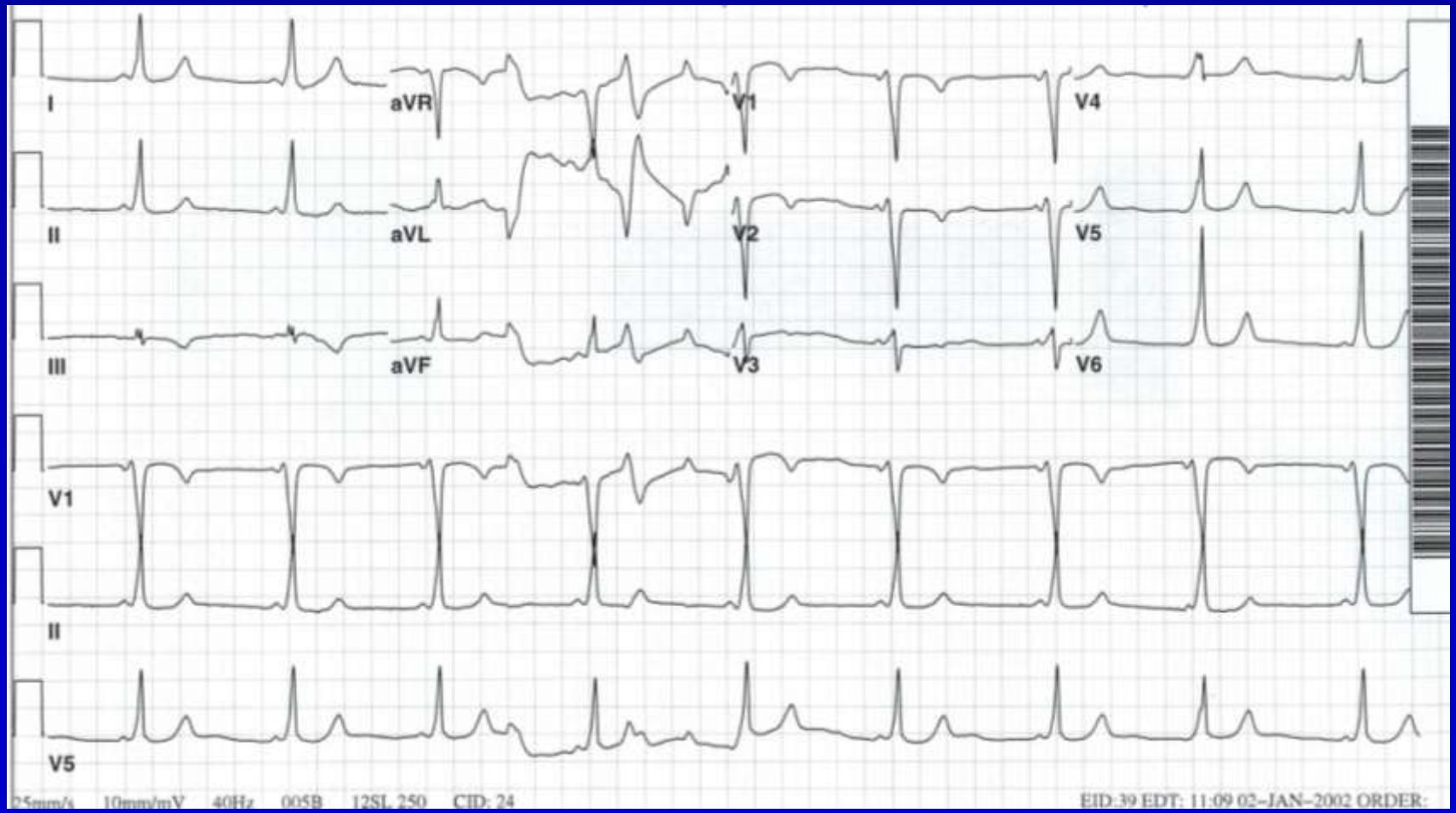
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V5
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V6
V1
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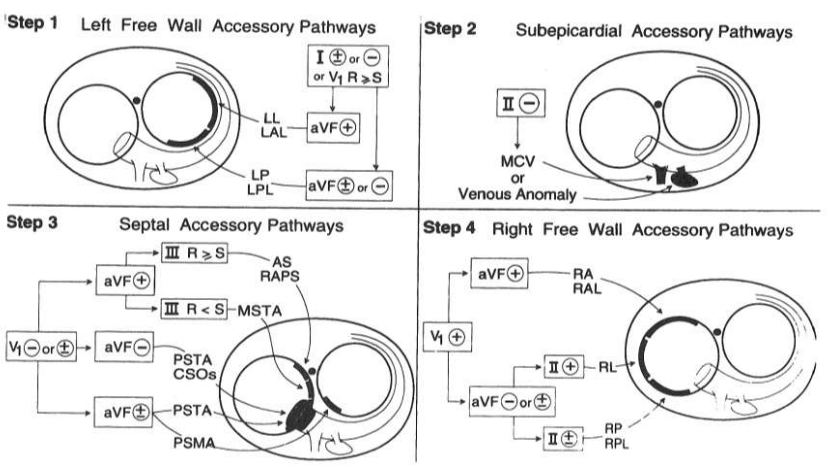
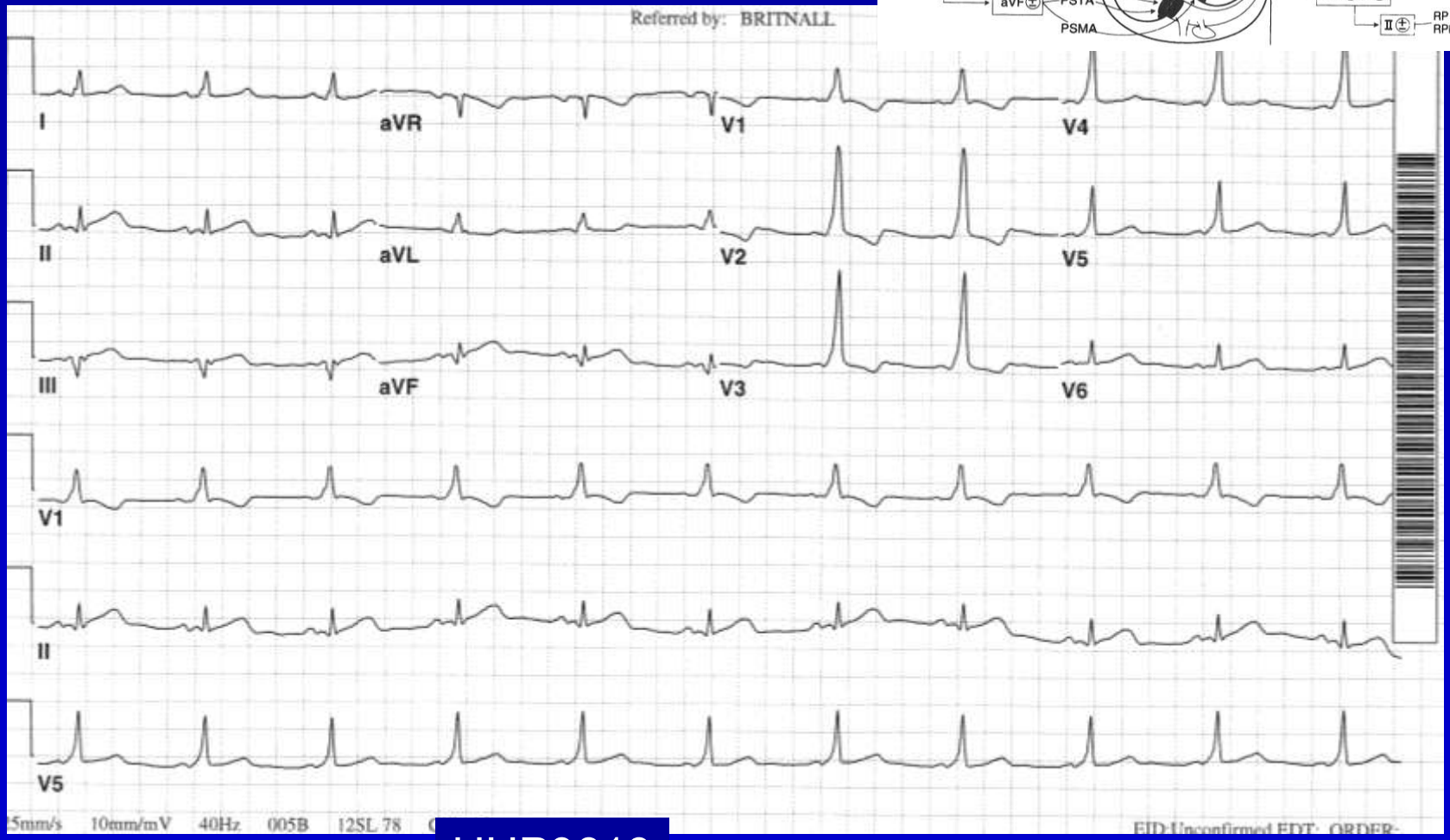
UHM7130

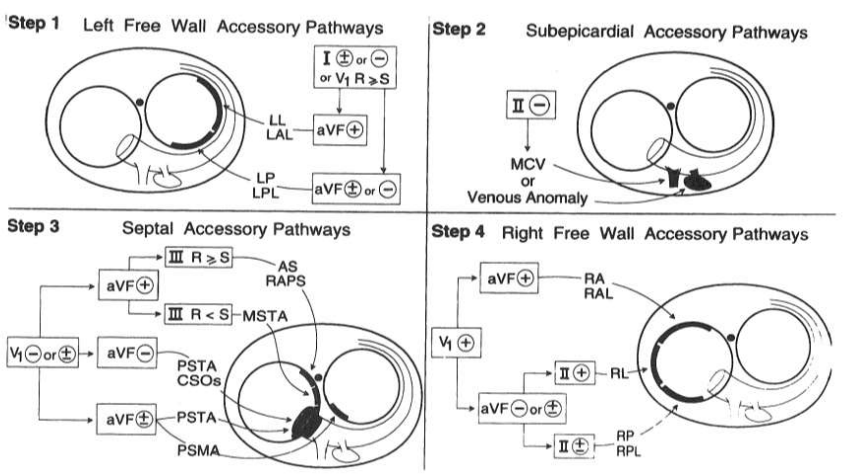
Left posterior or posterolateral

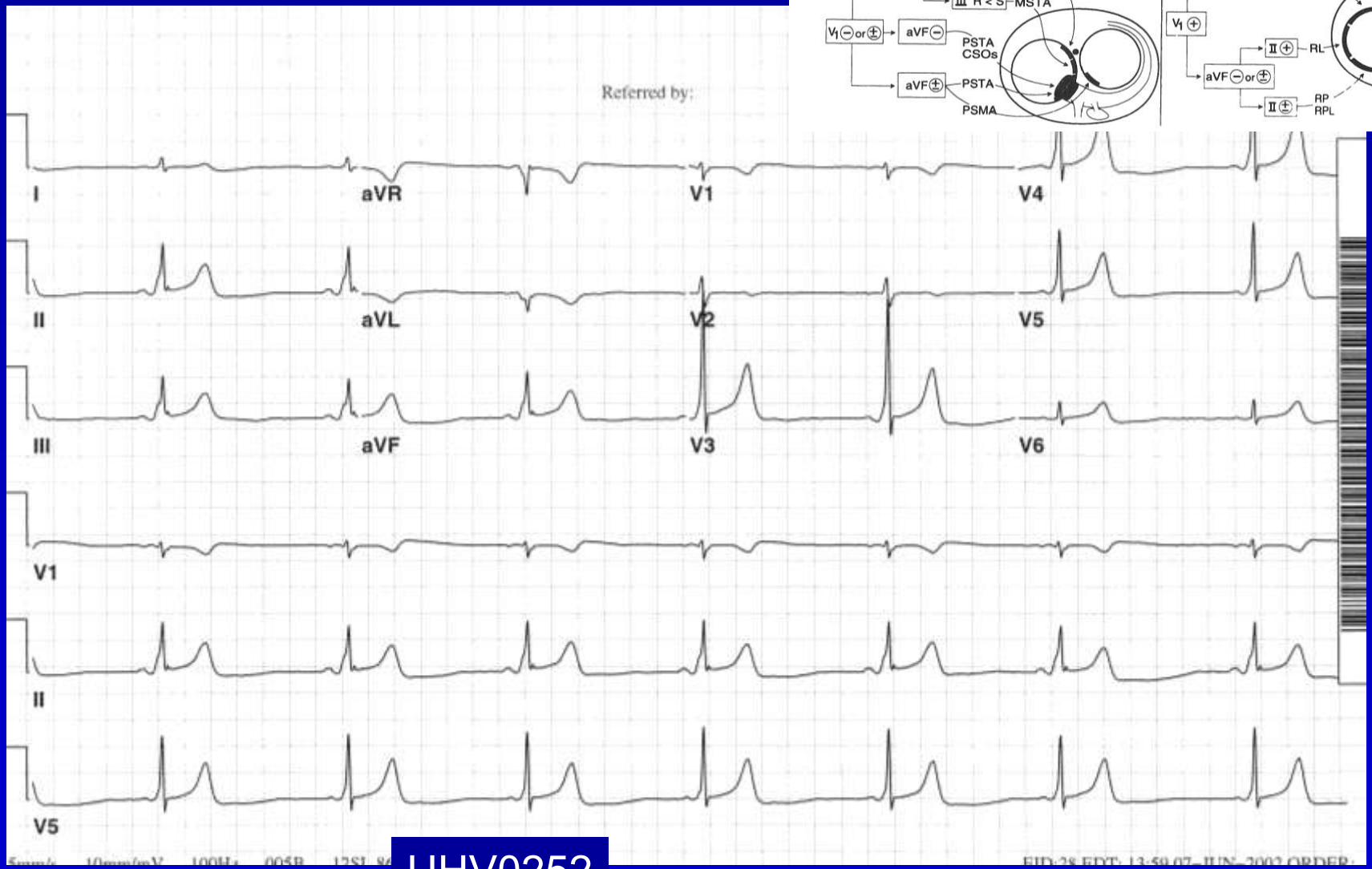




UHP9613

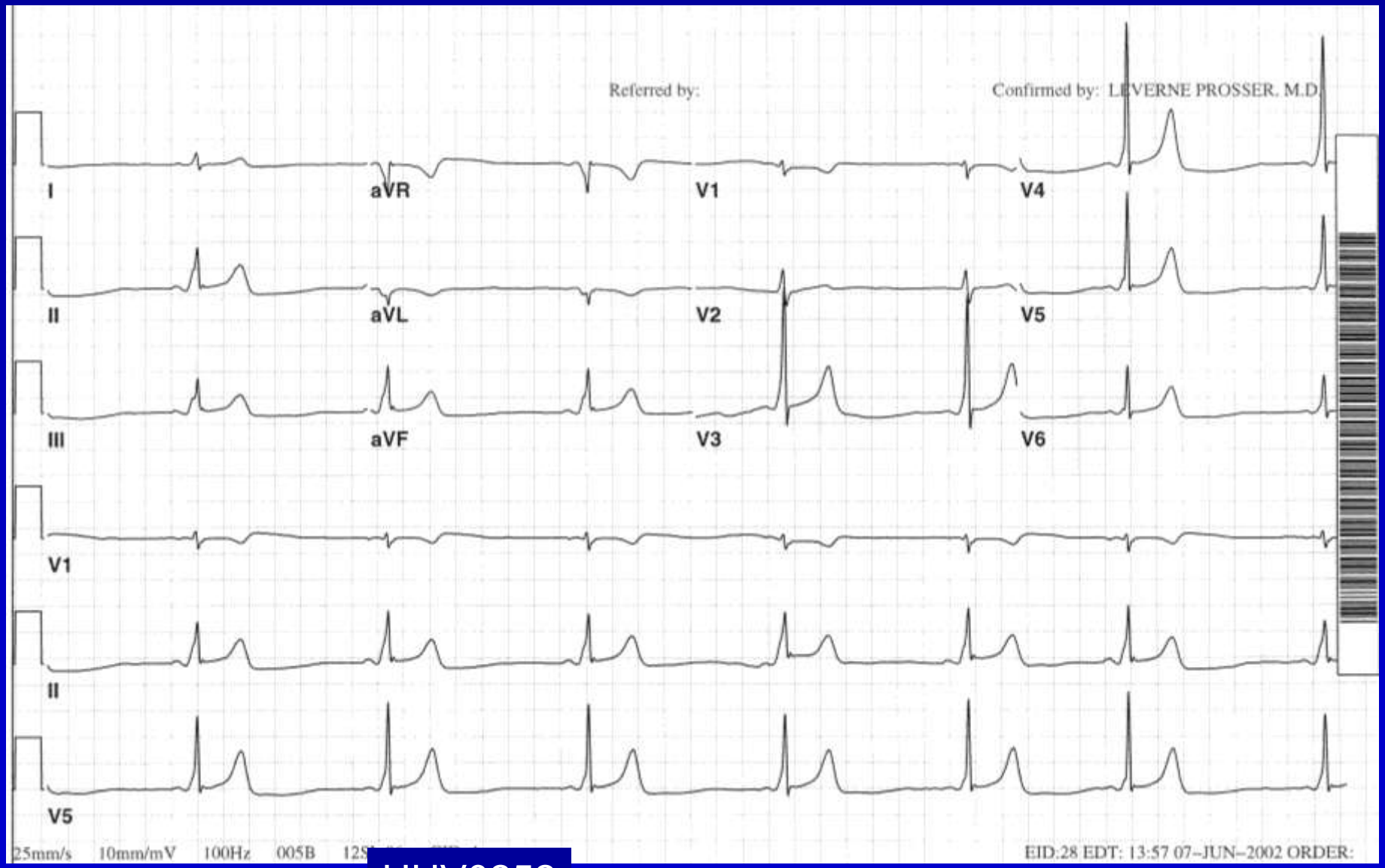
Left anterolateral or lateral





UHV0252

Left anterolateral or lateral



UHV0252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V2
V5
III
aVF
V3
V6
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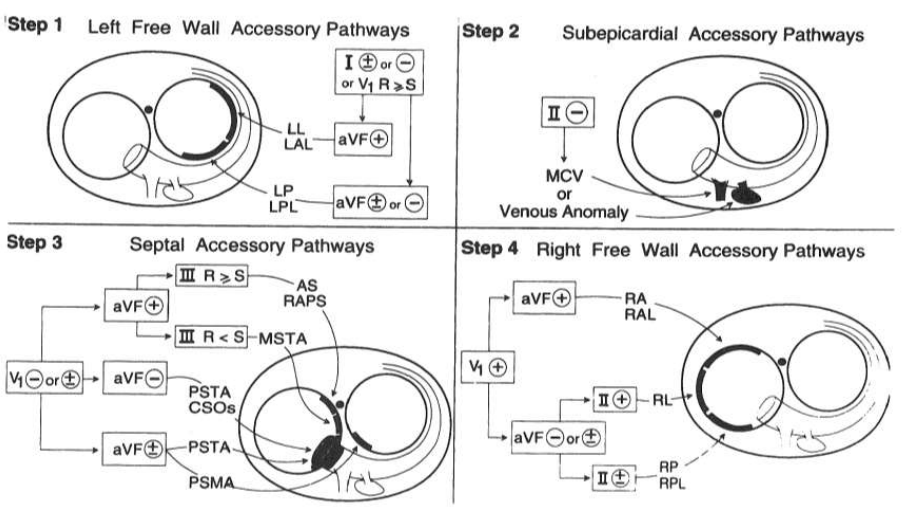
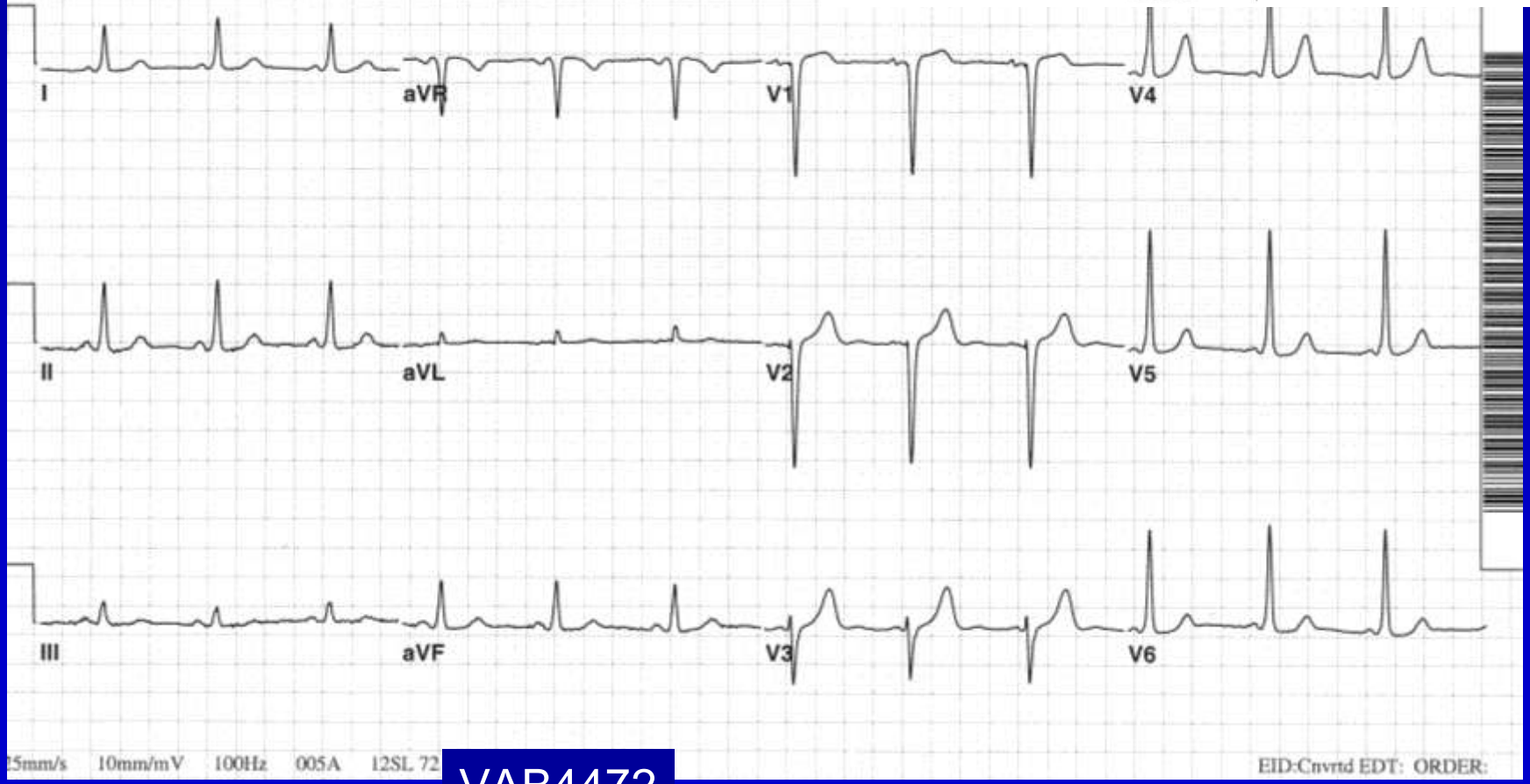
VAB4472

Right anteroseptal or anterior paraseptal





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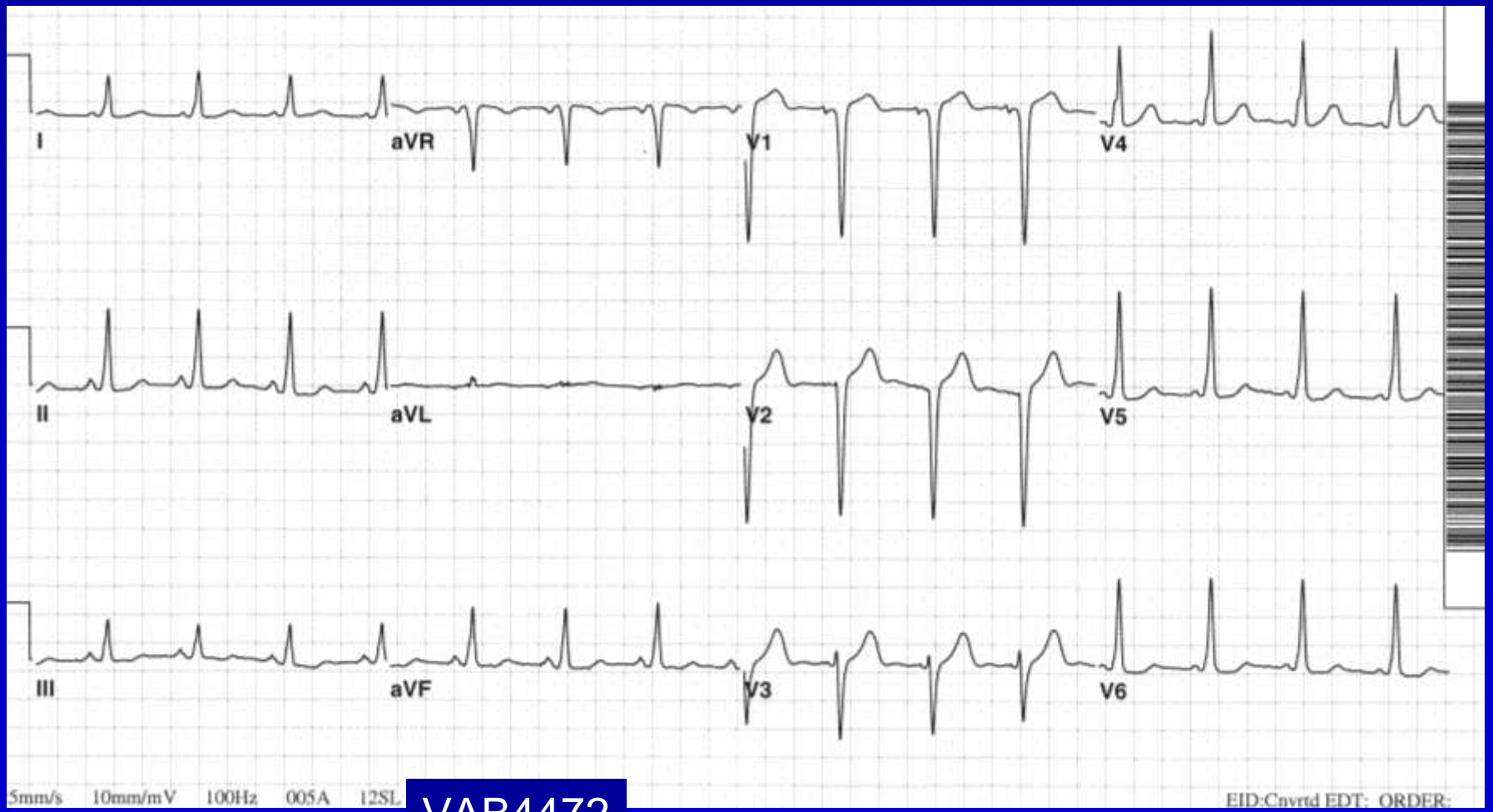
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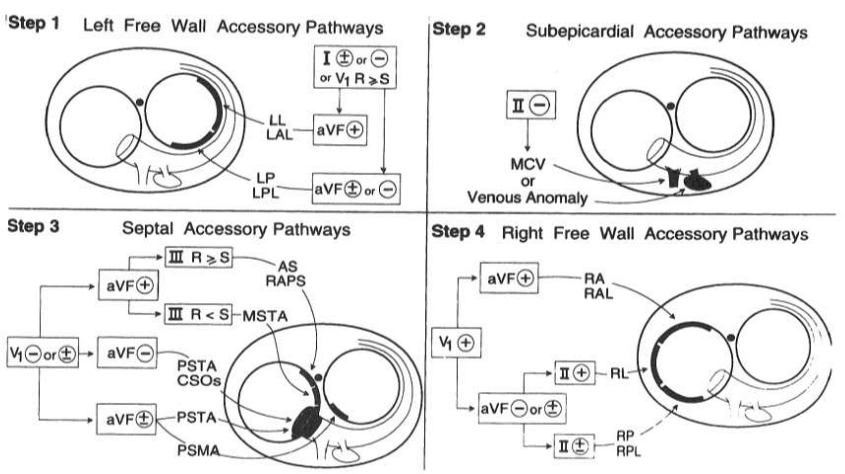
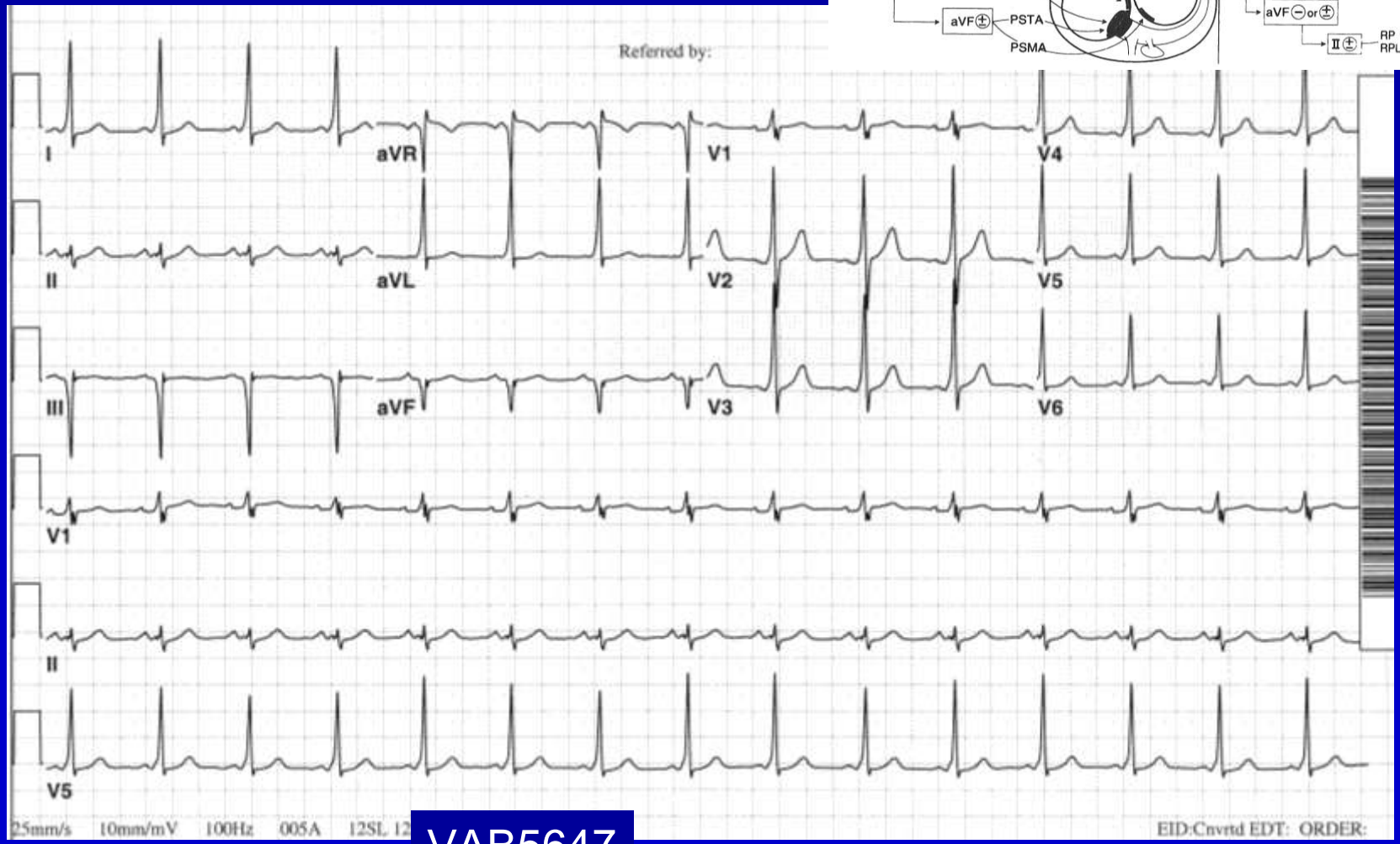
Left posterior





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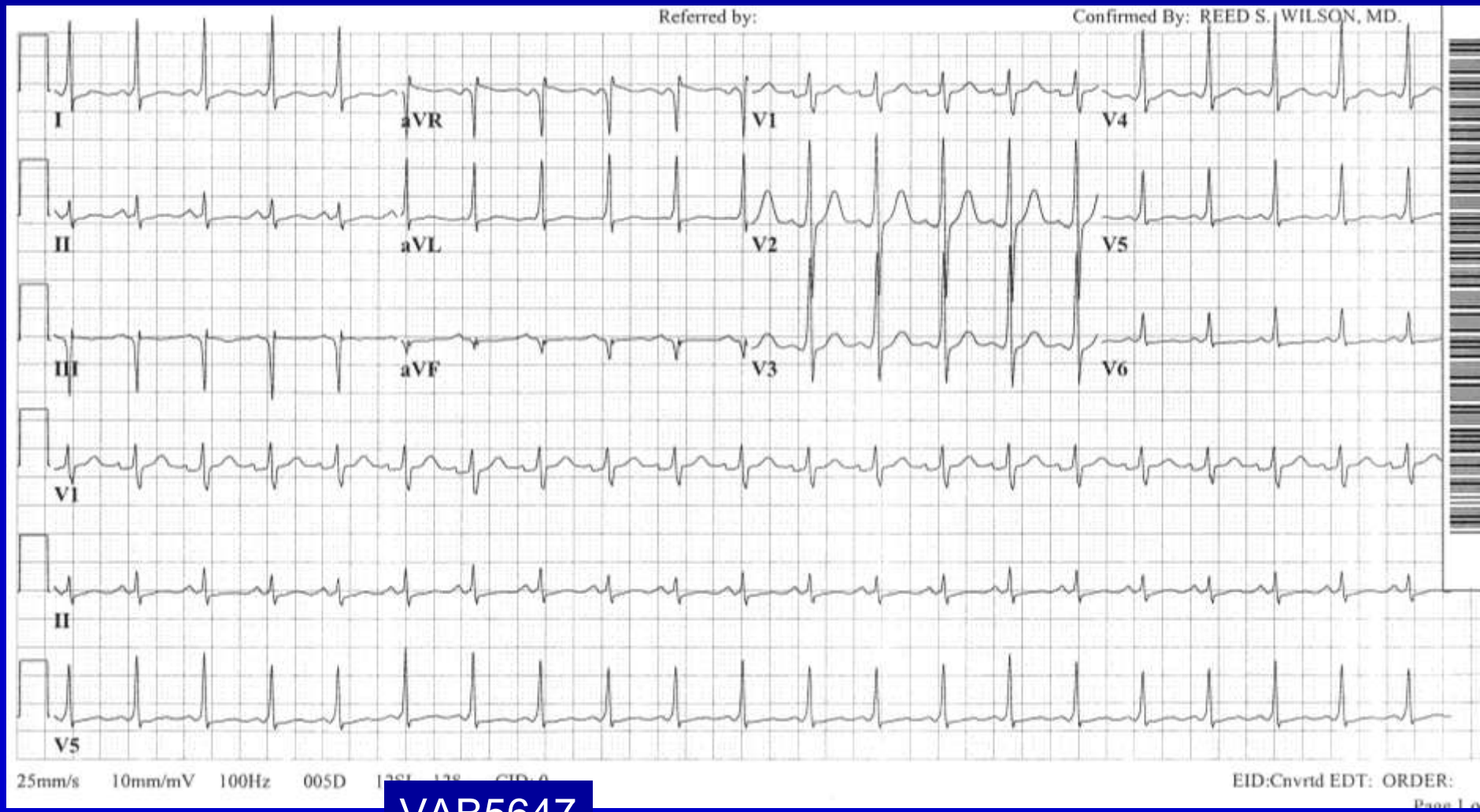
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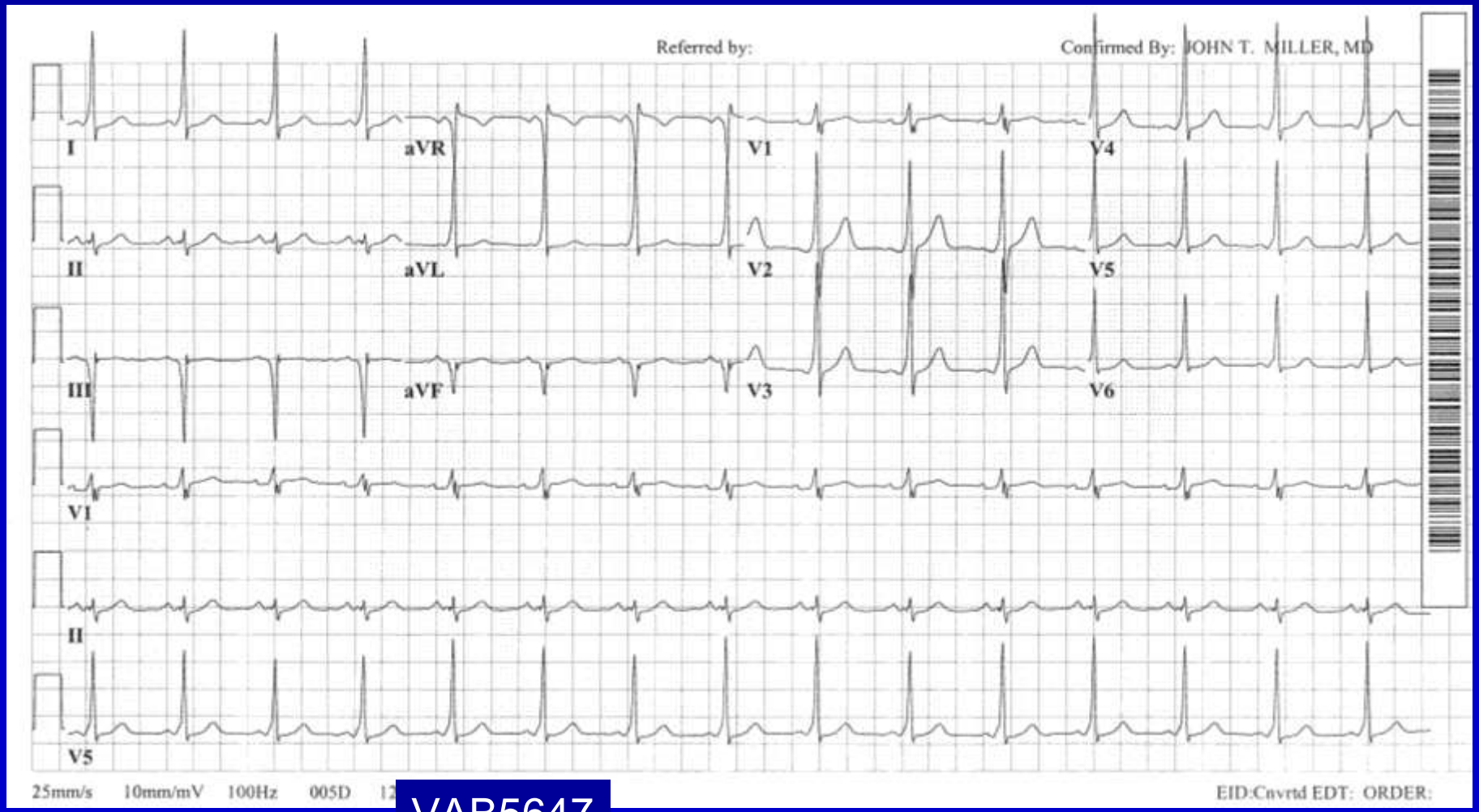
Confirmed By: REED S. WILSON, MD.
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s
10mm/mV
100Hz
005D
EID:Cnvrtd EDT: ORDER:

VAB5647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Confirmed By: JOHN T. MILLER, MD
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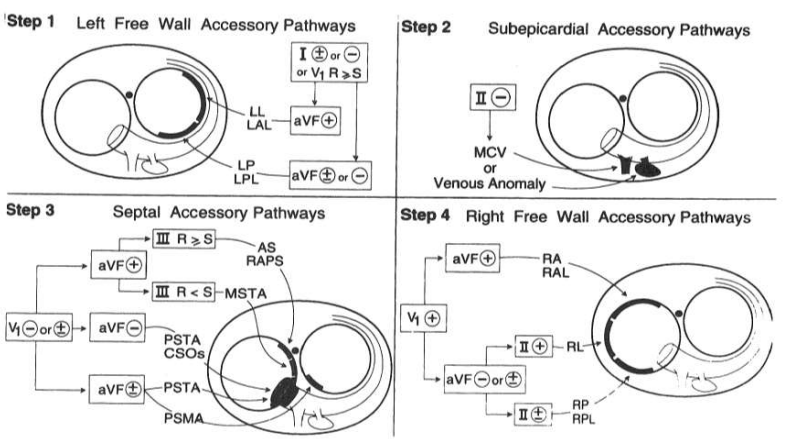
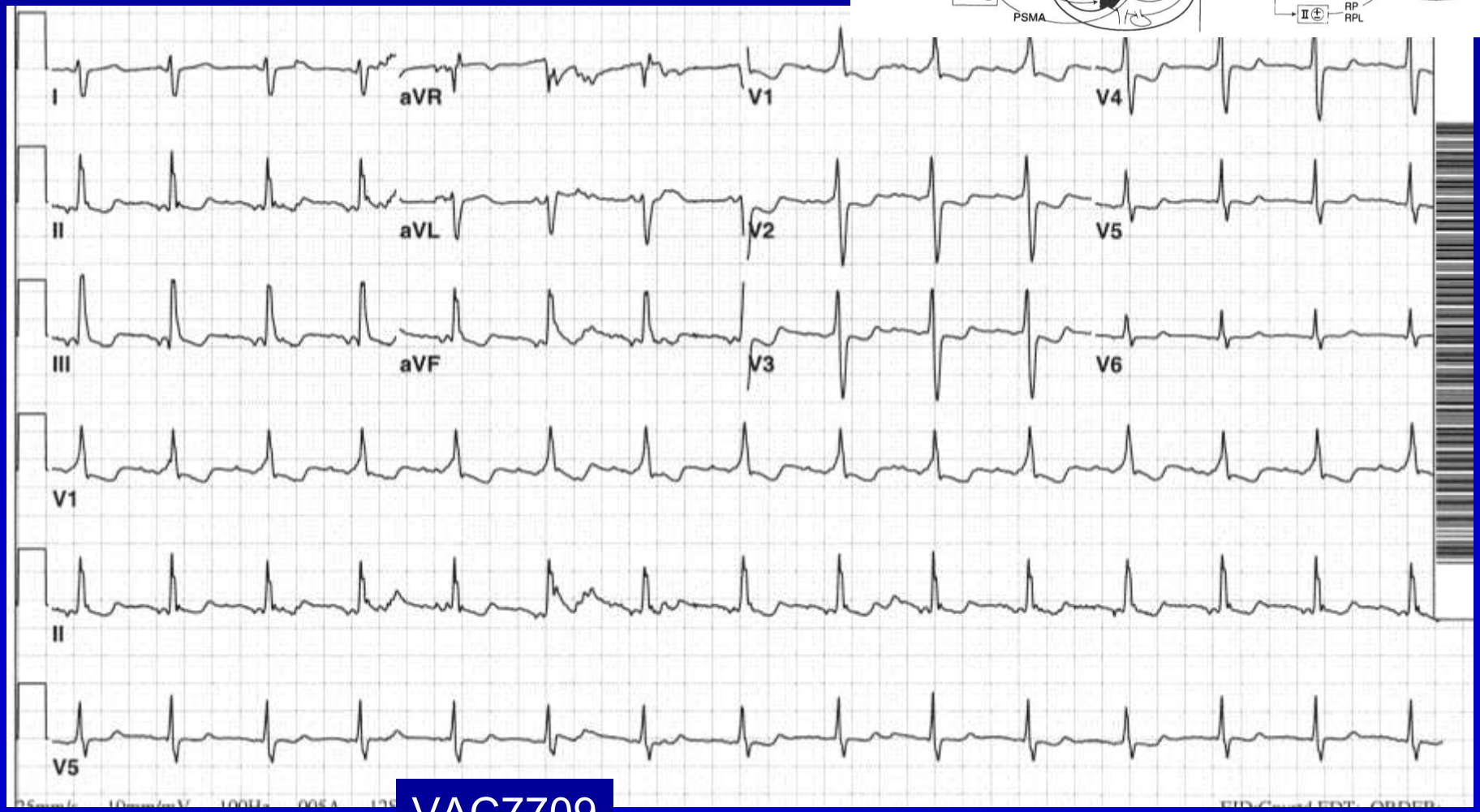
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s
10mm/mV
100Hz
005D
EID:Cnvrtd EDT: ORDER:

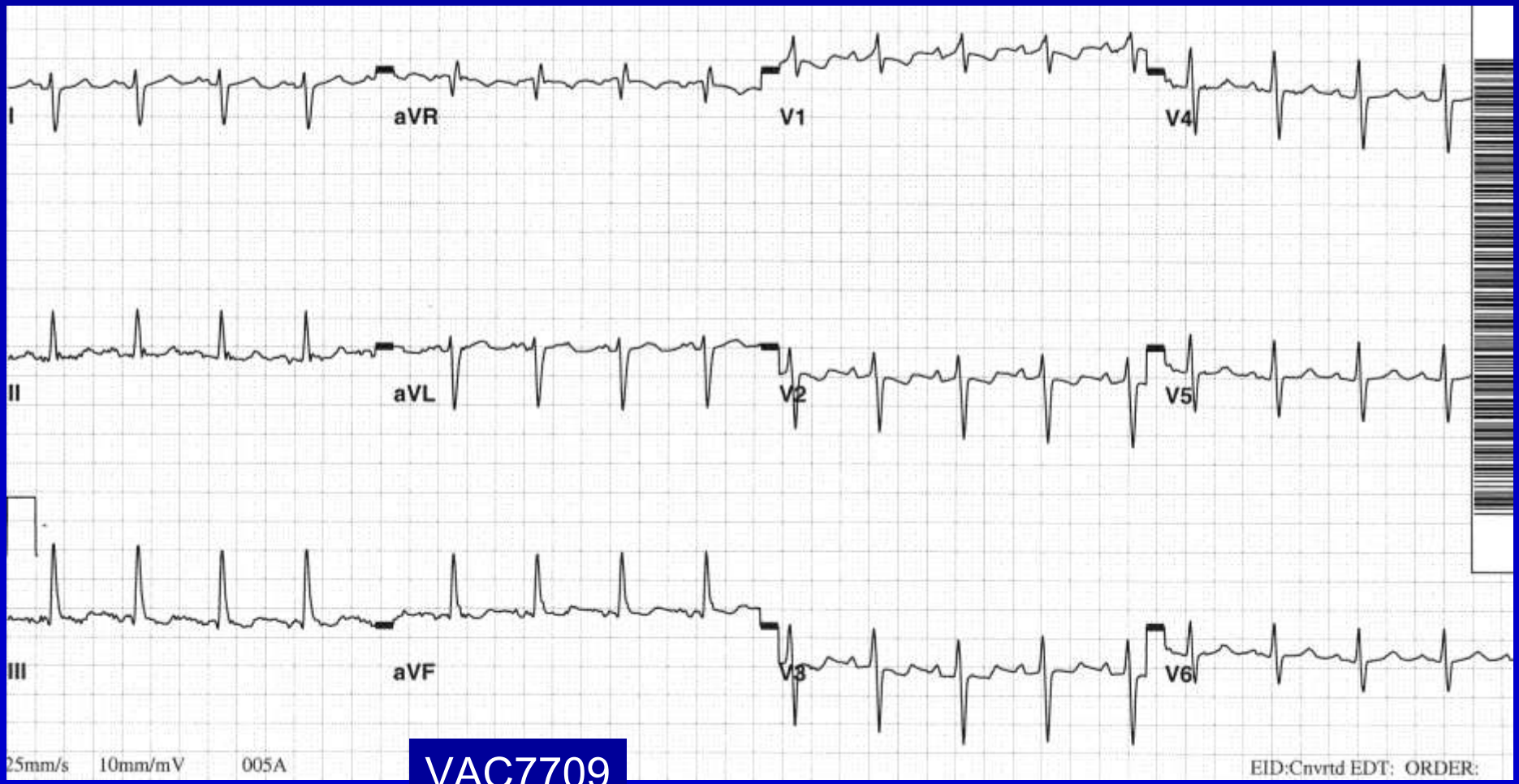
VAB5647

Left lateral



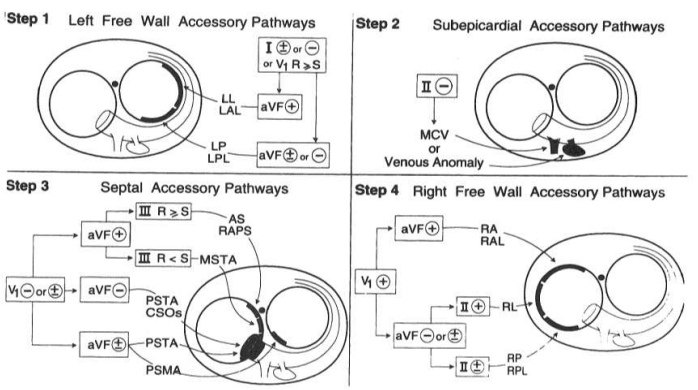


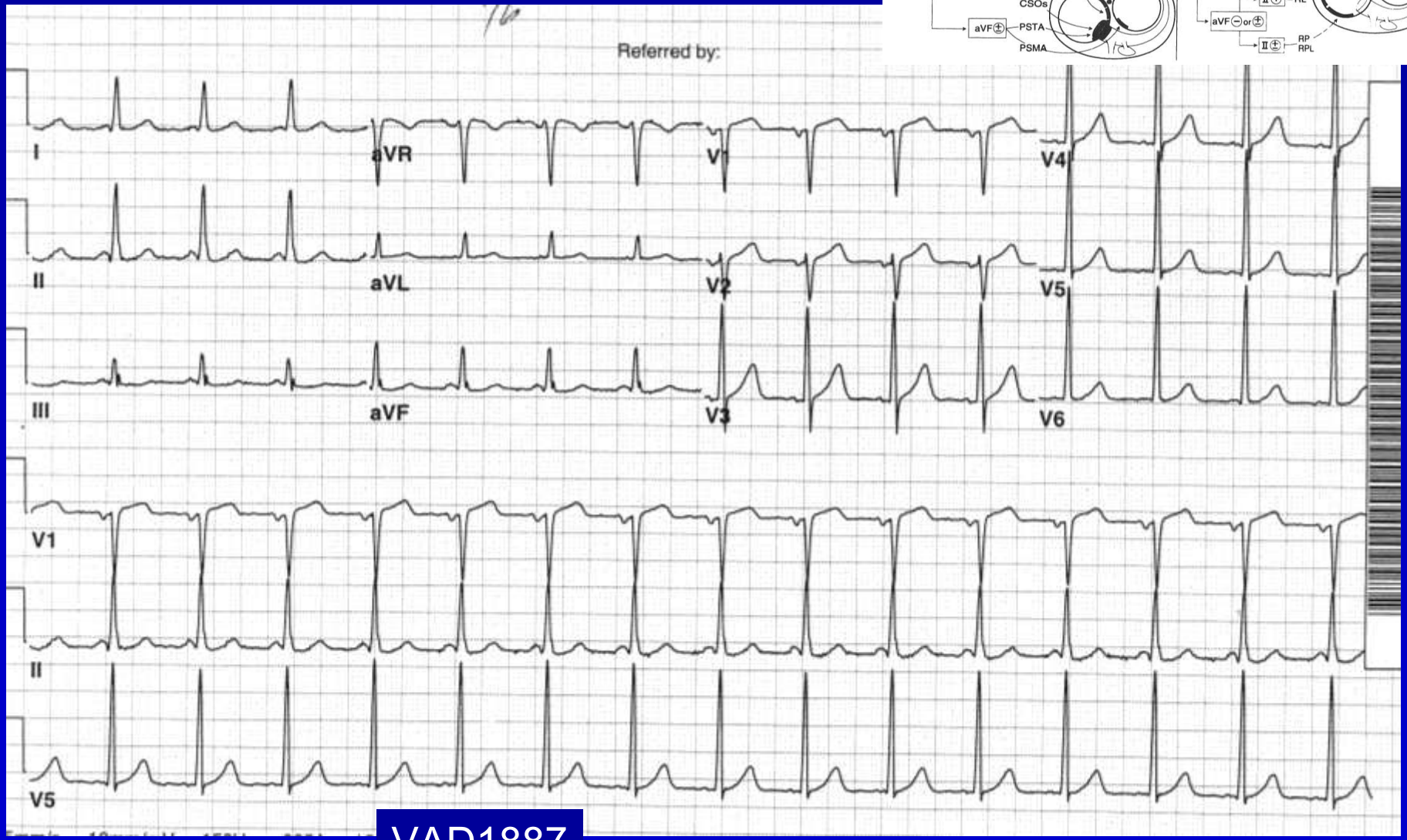
VAC7709


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
25mm/s
10mm/mV
005A
EID:Cnvrtd EDT: ORDER:

VAC7709

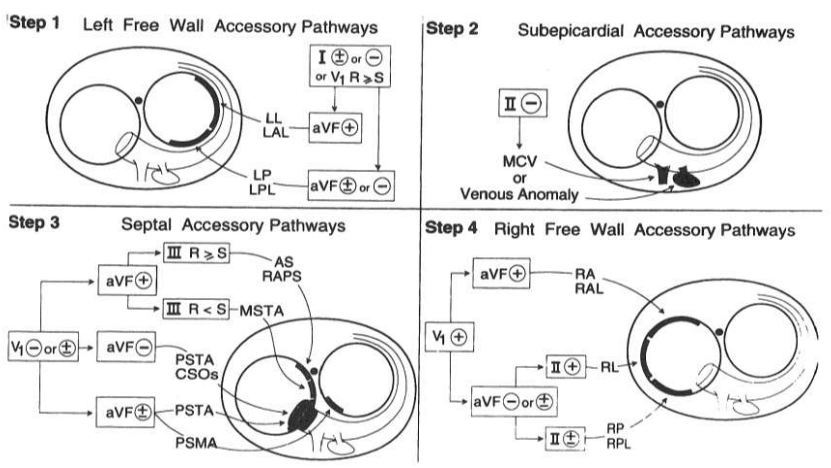
No delta wave – merely enhanced AV node conduction (LGL)

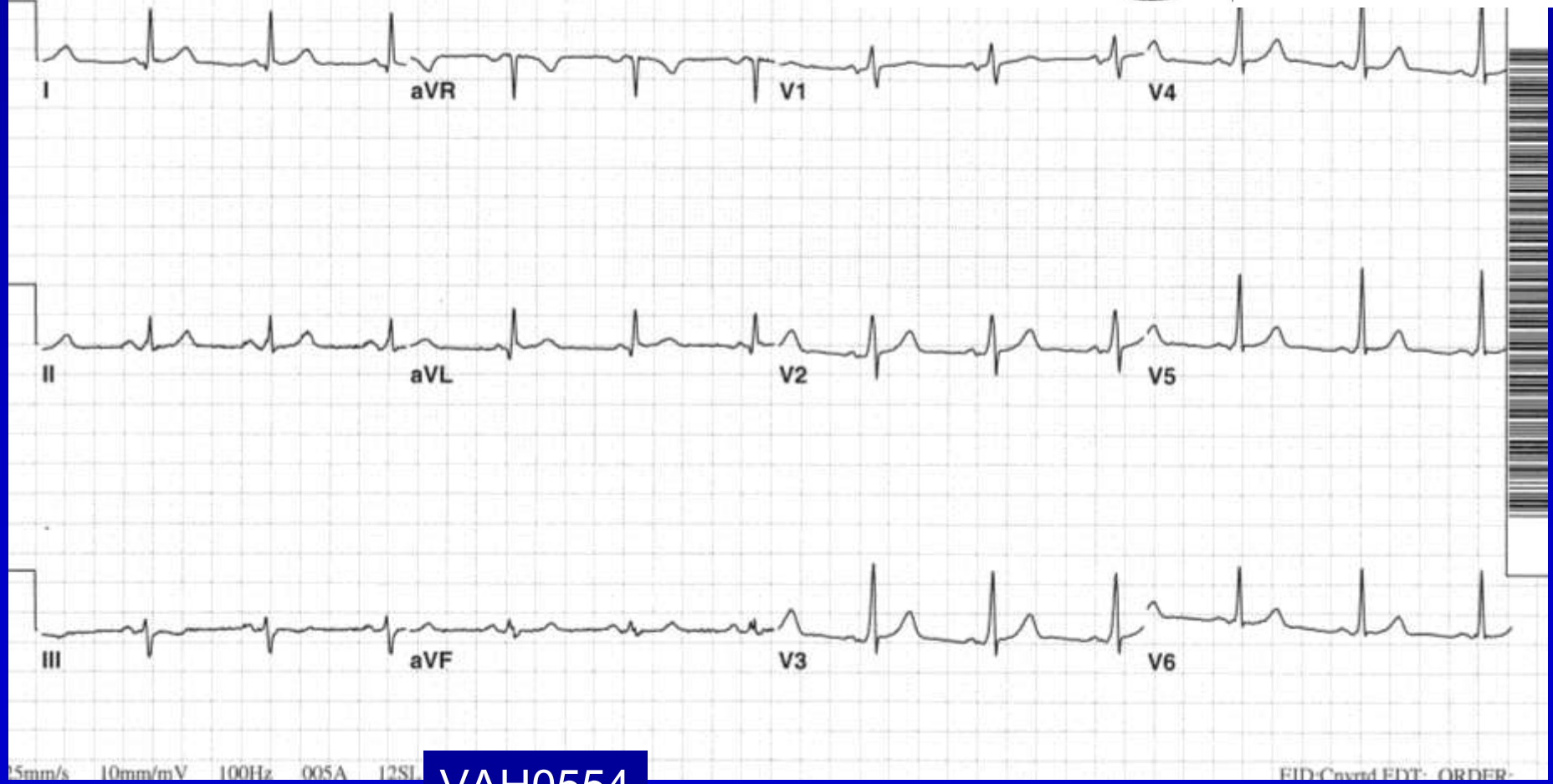




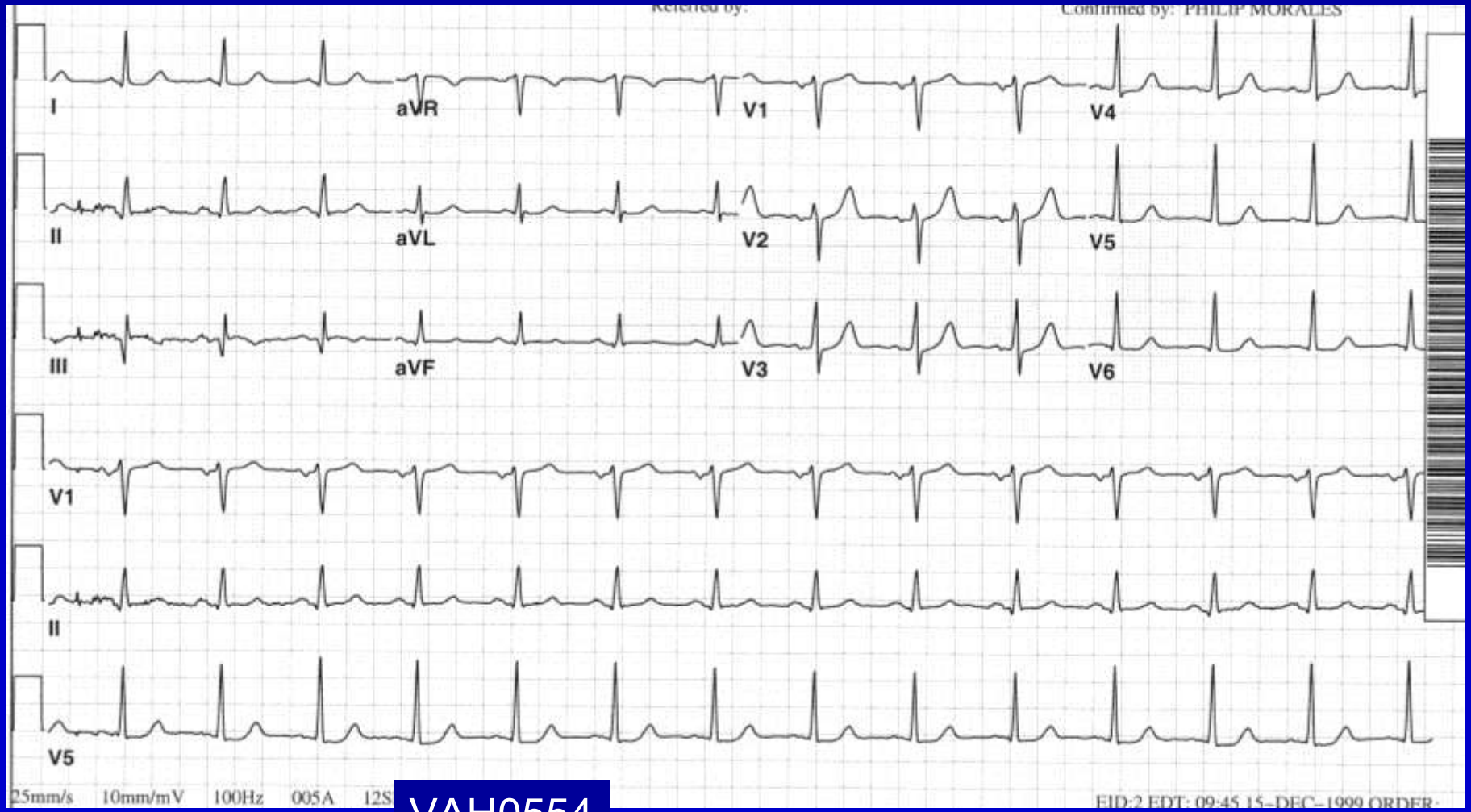
VAD1887

Not very pre-excited, left lateral





VAH0554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V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
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V5
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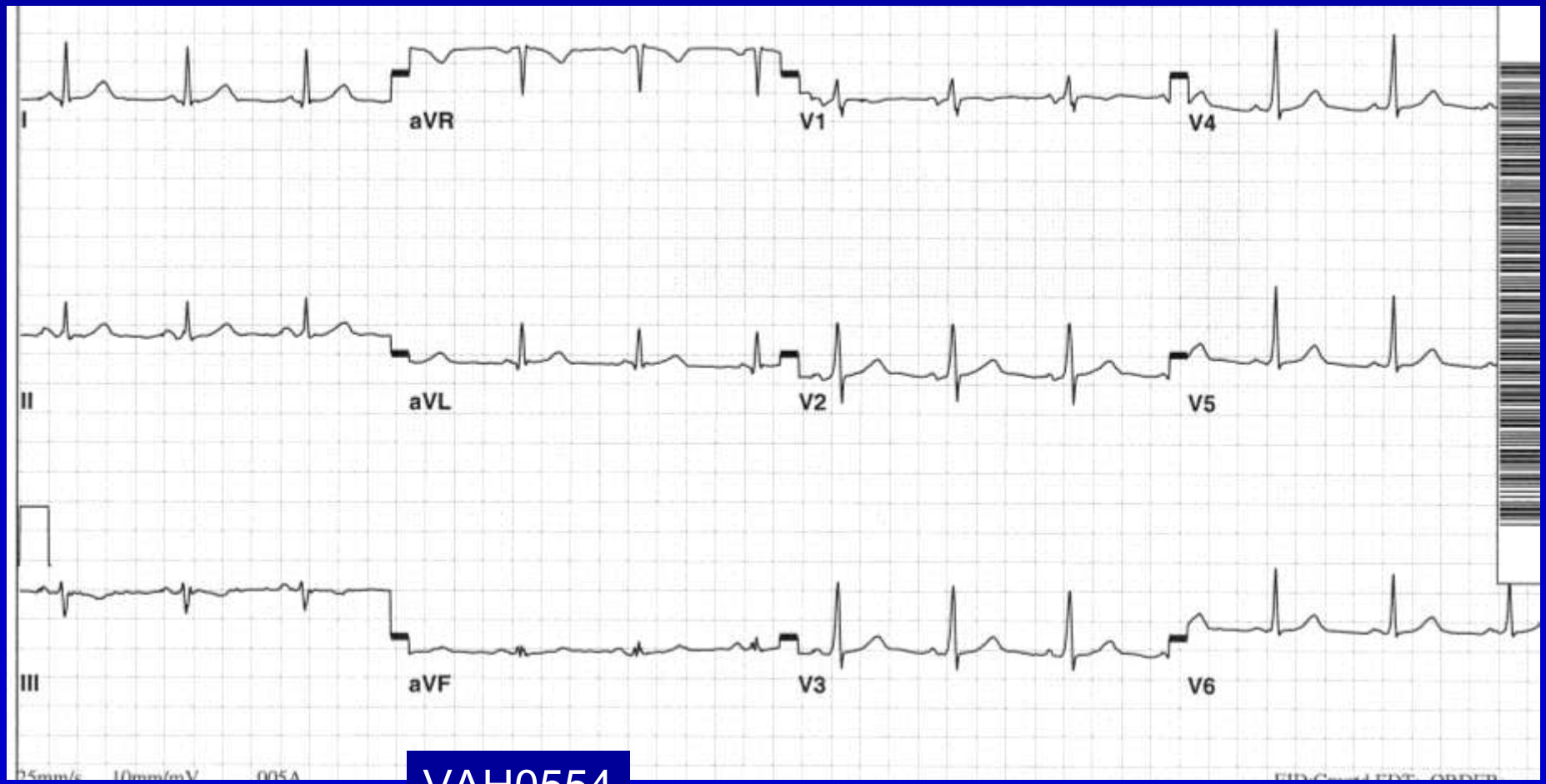
10mm/mV
100Hz
005A
12S
EID:2 EDT: 09:45 15-DEC-1999 ORDER:

VAH0554



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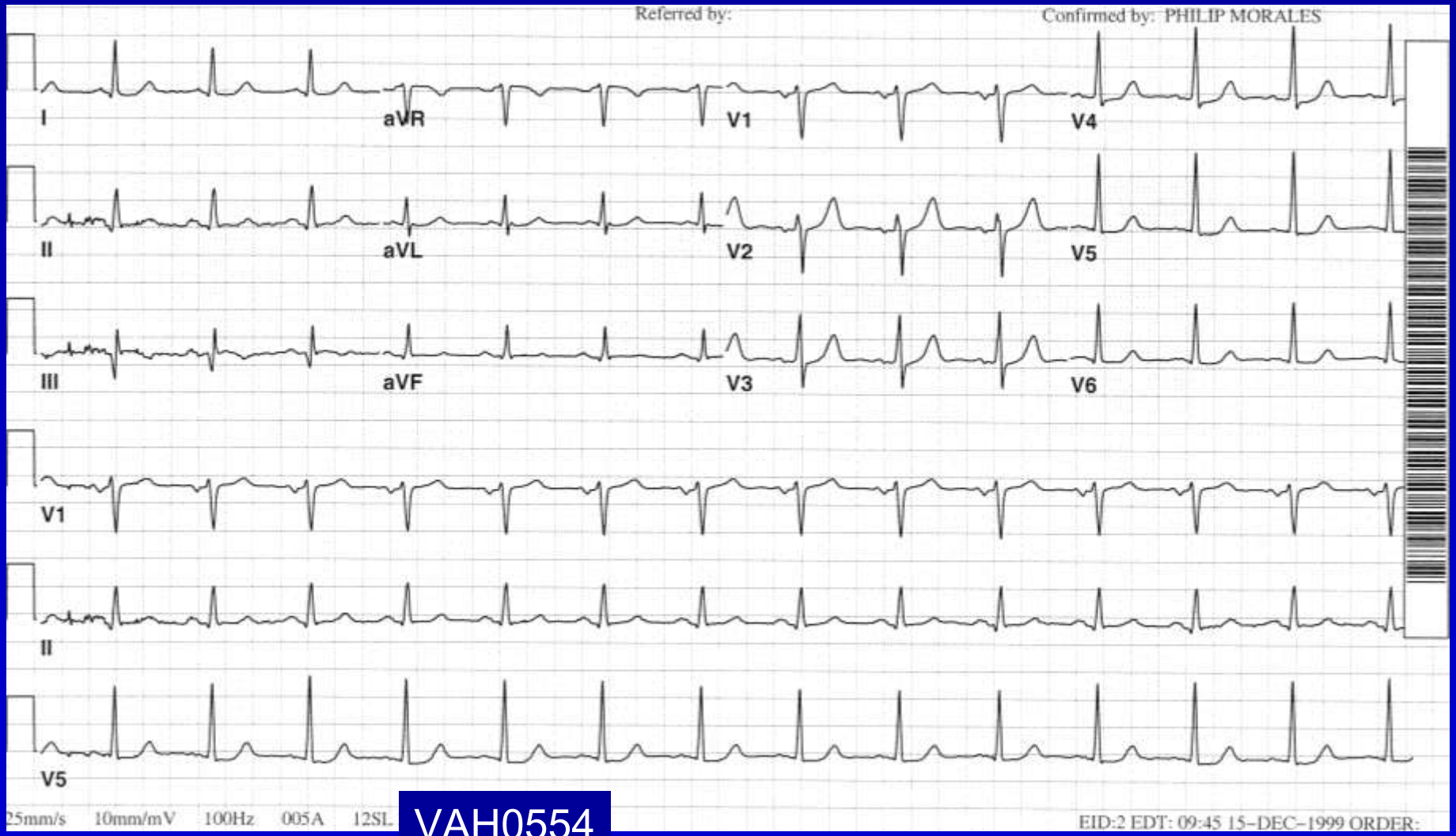
Referred by:
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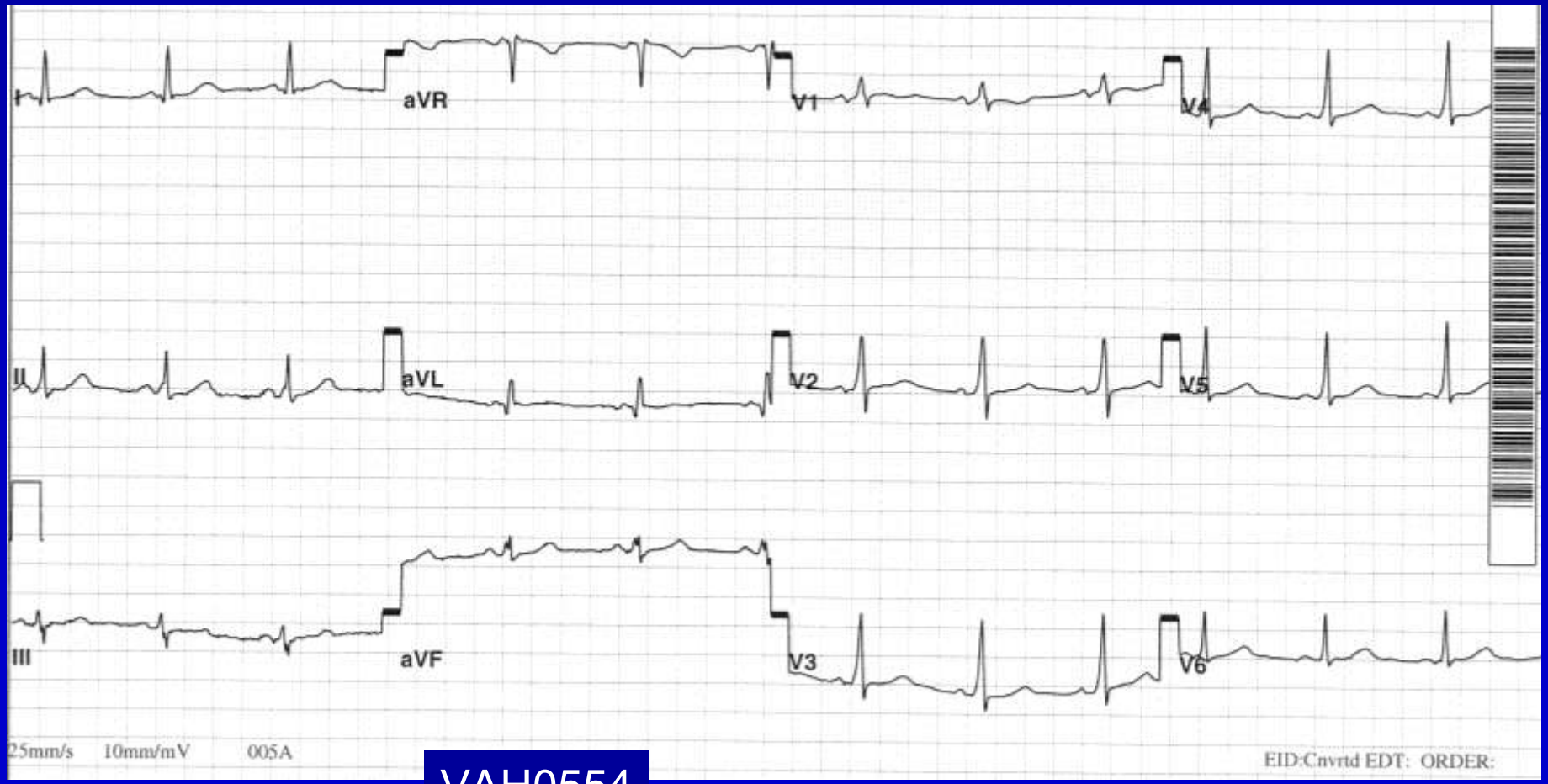
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s
10mm/mV
100Hz
005A
12SL
EID:2 EDT: 09:45 15-DEC-1999 ORDER:

VAH0554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V1
V4
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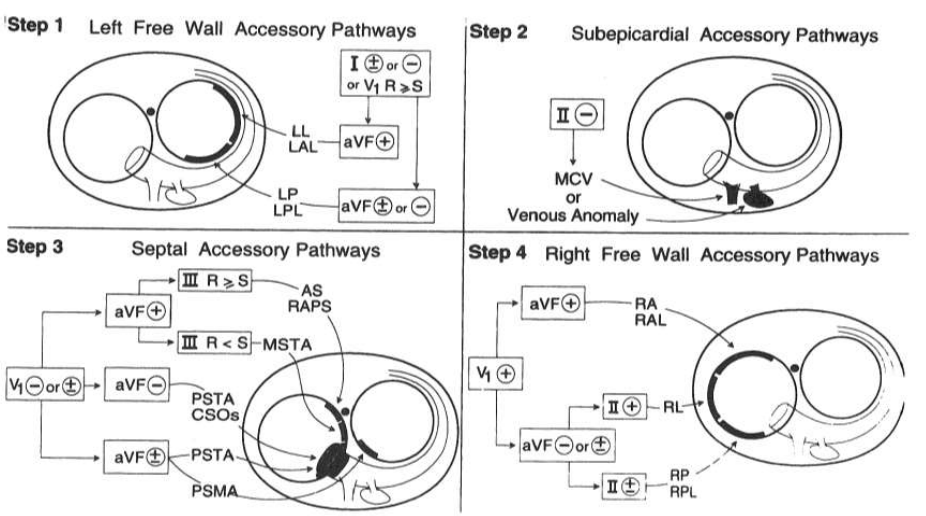
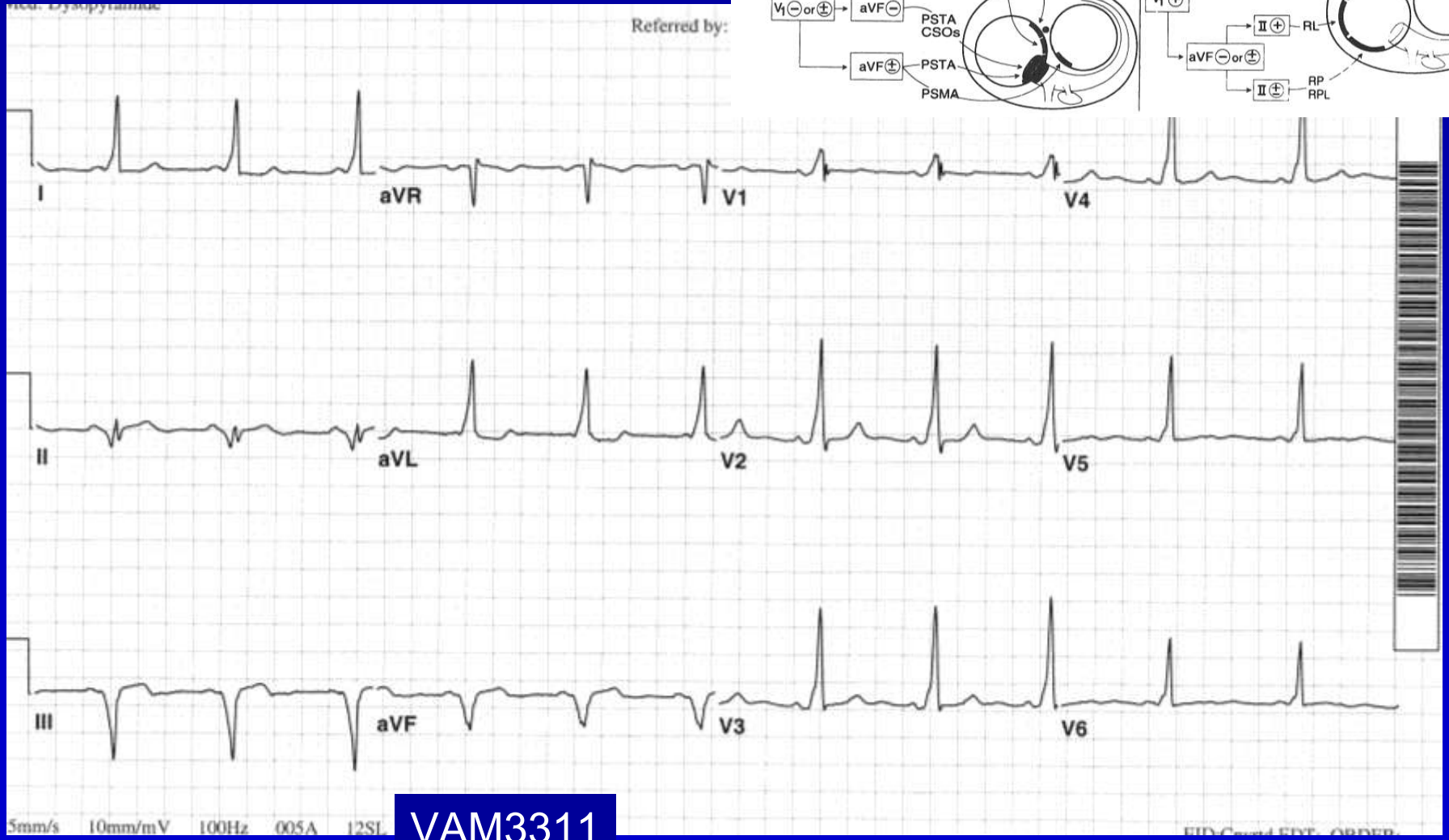
aVL
V2
V5
III
aVF
V3
V6
25mm/s
10mm/mV
005A
EID:Cnvrtd EDT: ORDER:

VAH0554

Left posterior or posterolateral





VAM3311

References

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Original Article, Cases V and VI

Fig. 13.—(Case V) Intraventricular block. The P-waves are normal and upright in all leads. The P-R interval is well under 0.1 second.

Fig. 14.—(Case VI) Intraventricular block. The P-waves are normal and upright in all leads. The P-R interval is well under 0.1 second.

Original Article

Fig. 15.—(Case VII) Left bundle-branch block. The P-waves are normal and upright in all leads. The P-R interval is well under 0.1 second.

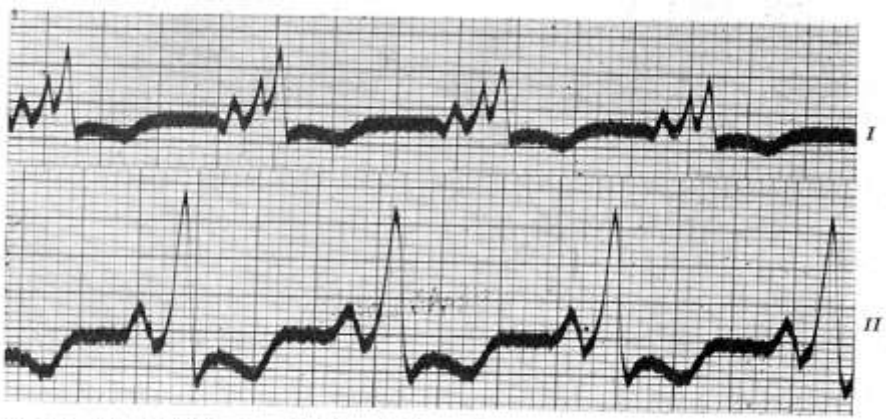

Fig. 18.—(Case IX) Intraventricular block. The P-R interval is 0.1 second. Time intervals = 0.2 and 0.04 seconds.

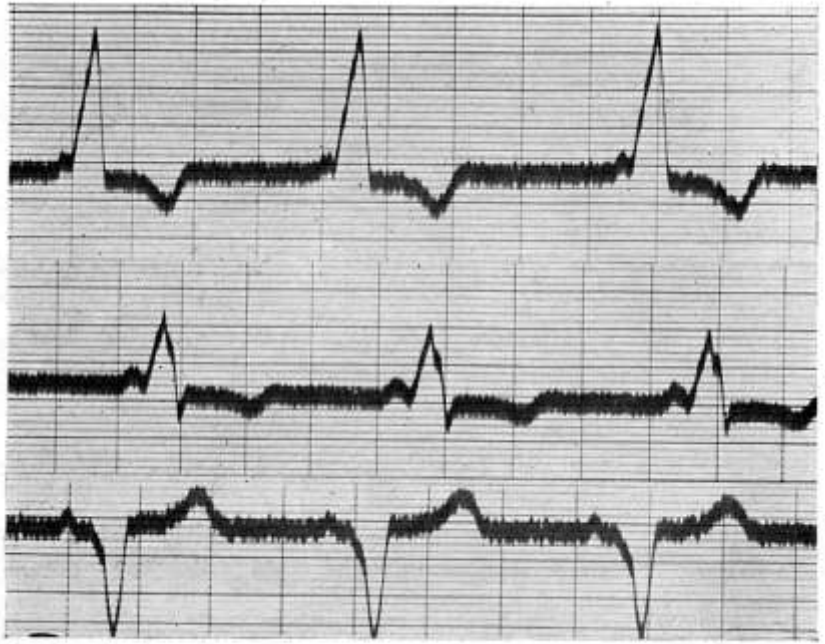

Fig. 16.—(Case VIII) Right bundle-branch block. The P-R interval is well under 0.1 second.

Fig. 17.—(Case VIII) Three years later. Normal physical curves. The P-R interval is 0.16 second. The P-waves are identical in Figs. 16 and 17.

Original Article

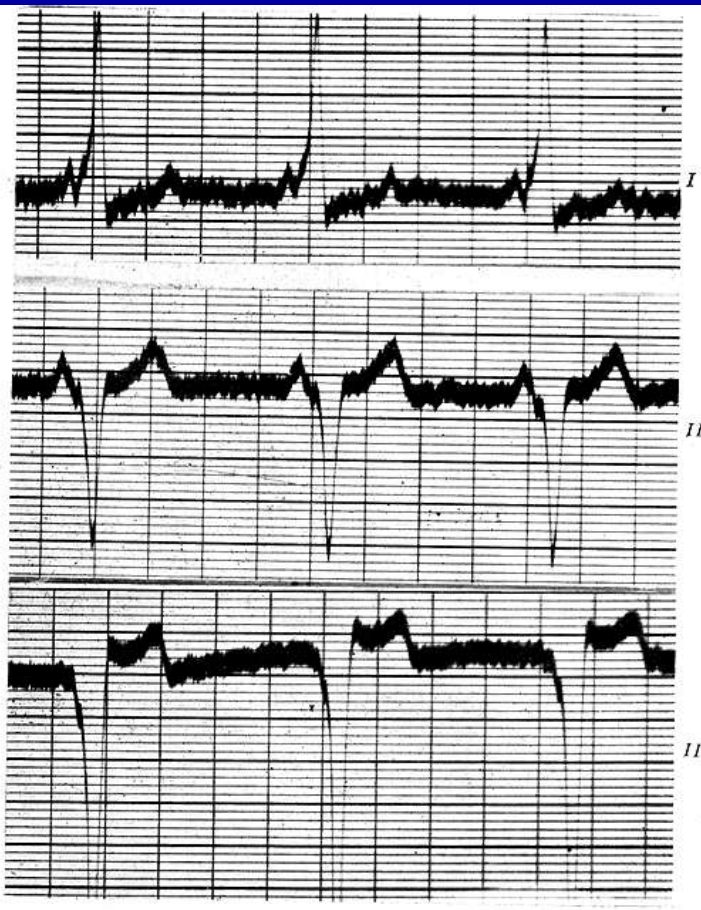

Fig. 19.—(Case X) Intraventricular block. The P-R interval is 0.1 second.

Fig. 20.—(Case XI) Intraventricular block. The P-R interval is less than 0.1 second.

Original Article

NOTE: In this paper bundle-branch block when mentioned is referred to according to the old nomenclature of right bundle-branch block for upright widened Q-R-S waves in Lead I and inverted widened Q-R-S waves in Lead III, and left bundle-branch block for inverted widened Q-R-S waves in Lead I, and upright widened Q-R-S waves in Lead III according to the newly revised nomenclature, which is probably correct, these designations would be changed, so that one should read "left bundle-branch block" for "right" and "right bundle-branch block" for "left" in this paper.

REFERENCES

1. Carter, E. P.: Clinical Observations on Defective Conduction in the Branches of the Auriculo-Ventricular Bundle. Arch. Int. Med., **13**, 803, 1914.
2. Cohn, A. E., and Lewis, T.: The Pathology of Bundle-Branch Lesions of the Heart. Proc. N. Y. Path. Soc., **14**, 207, 1914.
3. Eppinger, H., und Rothberger, J.: Zur Analyse des Elektrokardiogramms. Wien Klin. Wchnschr., **22**, 1091, 1909.
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5. Eppinger, H., und Stoerk, O.: Zur Klinik des Elektrokardiogramms. Ztschr. f. klin. Med., **71**, 157, 1910.
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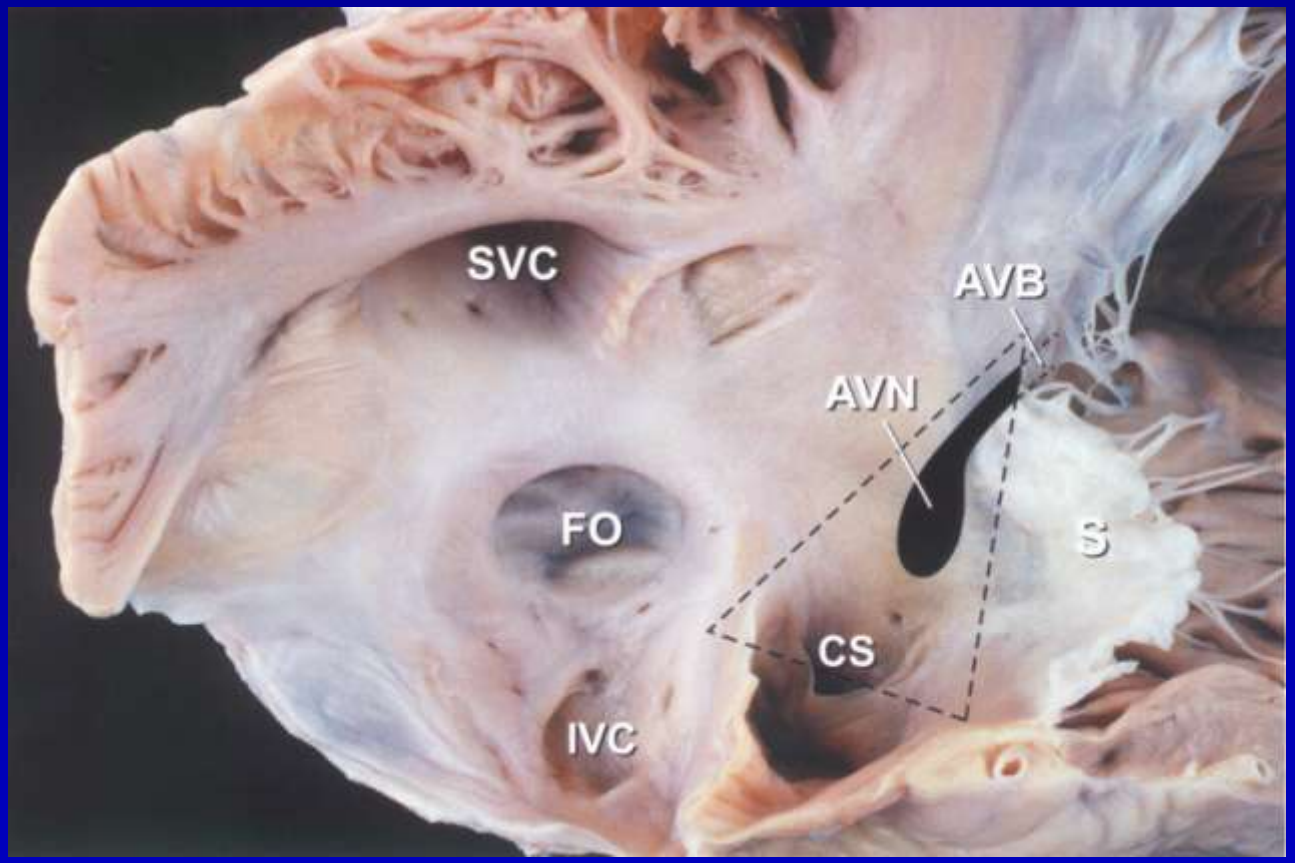
AV Node Land-marks

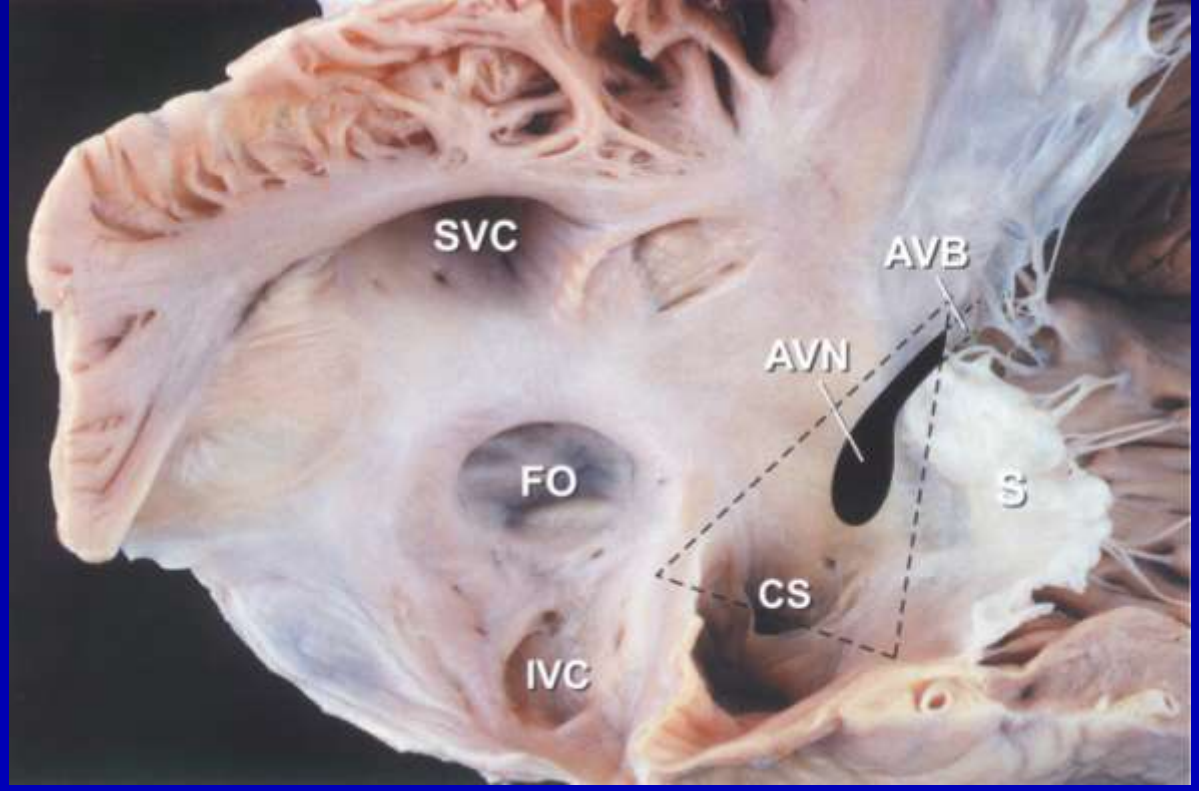


- Anatomical landmarks of the triangle of Koch. This triangle is delimited by the tendon of Todaro superiorly, which is the fibrous commissure of the flap guarding the openings of the inferior vena cava and coronary sinus, by the attachment of the septal leaflet of the tricuspid valve inferiorly, and by the mouth of the coronary sinus at the base.

Tendon of Todaro is absent in about 2/3 of hearts; it originates in the central fibrous body and passes through the atrial septum to continue with the eustachian valve ..

Braunwald, 2005, p.656; Hurst, 2004, p.83

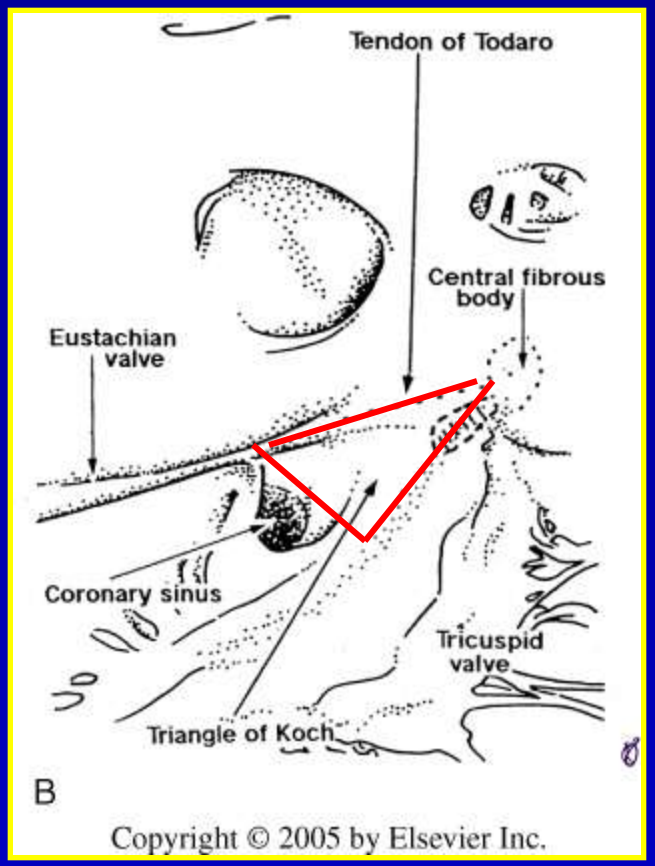
AV Node Land-marks

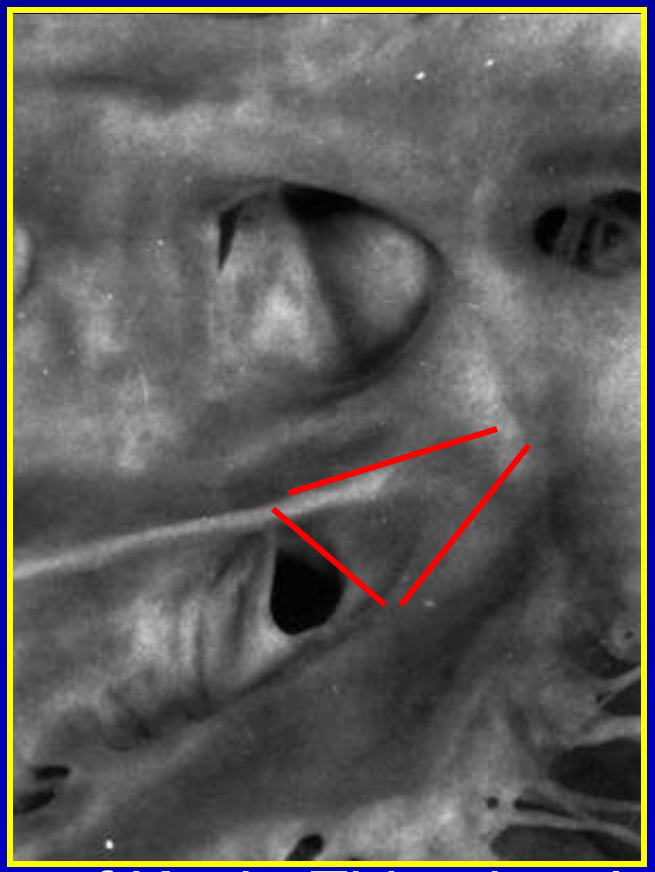


- The compact portion of the AVN becomes the penetrating portion of the His bundle at the point where it enters the central fibrous body
  - In 85-90% of human hearts the arterial supply to the AVN is from the RCA originating at the posterior intersection of the AV and interventricular grooves (crux)
- The Bundle of His (penetrating portion of the AV bundle) continues from the central fibrous body through the annulus fibrosis and penetrates the membranous septum

Braunwald, 2005, p.656; Hurst, 2004, p.83.

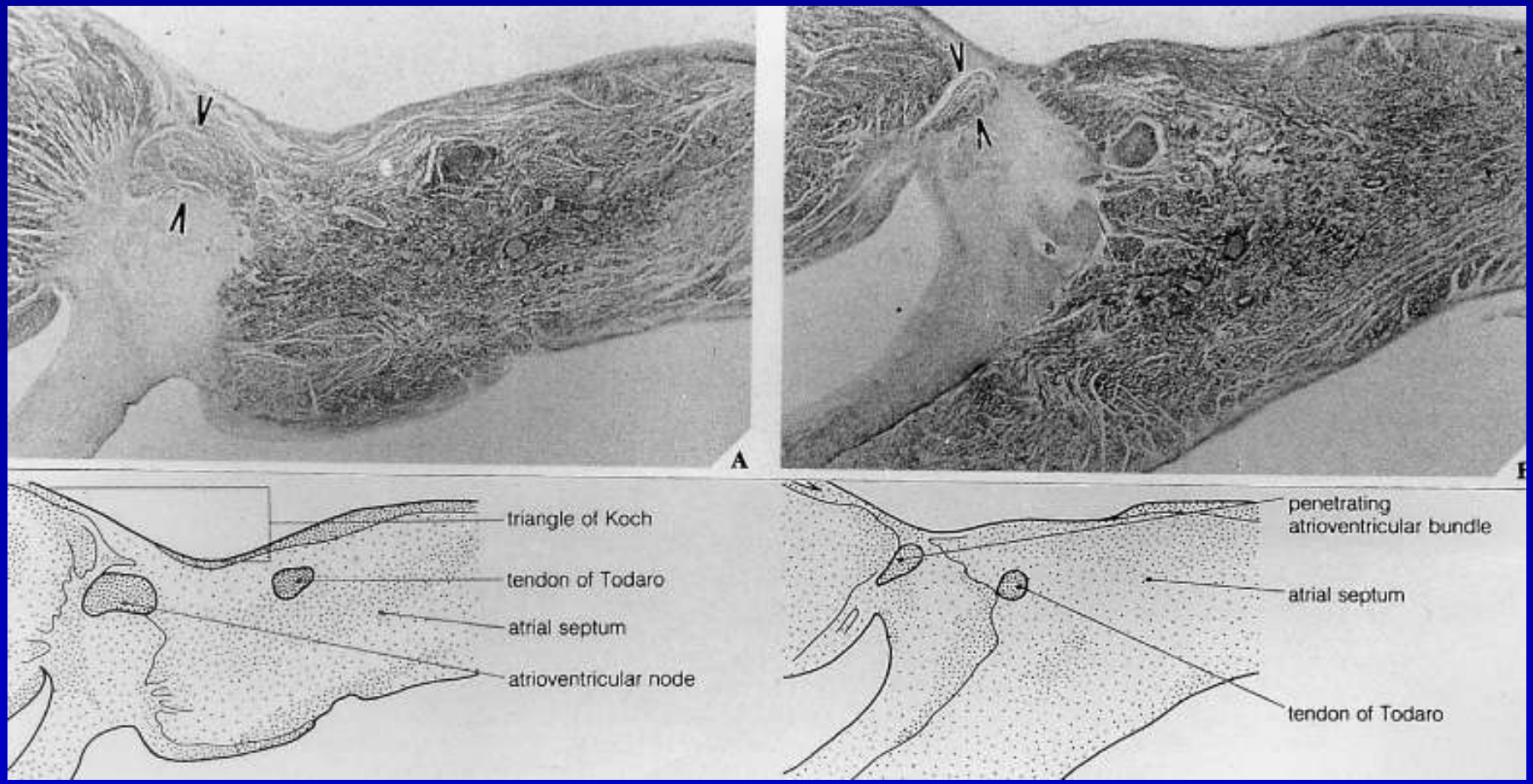
AV Node Land-marks





- Anatomical landmarks of the triangle of Koch. This triangle is delimited by the tendon of Todaro superiorly, which is the fibrous commissure of the flap guarding the openings of the inferior vena cava and coronary sinus, by the attachment of the septal leaflet of the tricuspid valve inferiorly, and by the mouth of the coronary sinus at the base. Stippled area adjacent to the central fibrous body is the approximate site of the compact atrioventricular node.

Braunwald, 2005, p.656.



- Sections through the atrioventricular (AV) junction show the position of the AV node (arrowhead) within the triangle of Koch **(A)** and the penetrating AV bundle of His (arrowheads) within the central fibrous body **(B)**.

Braunwald, 2005, p.657.