

Bioengineering 6460

Clinical EP Studies

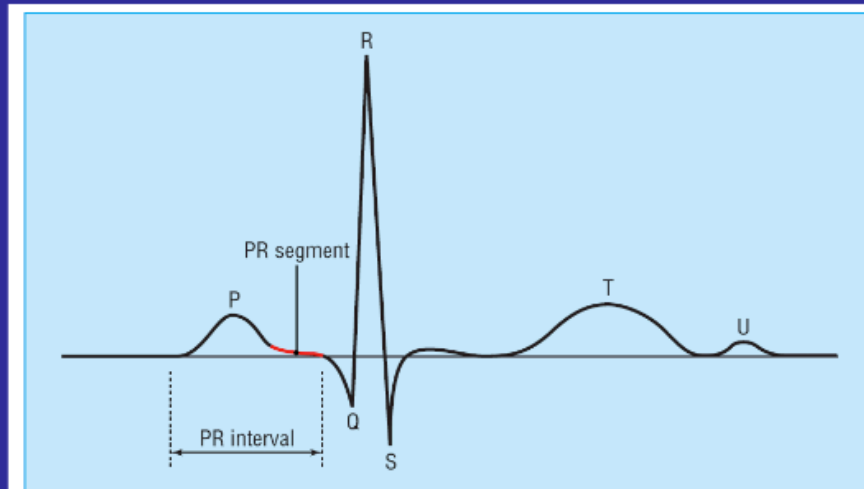
Ravi Ranjan

Division of Cardiology

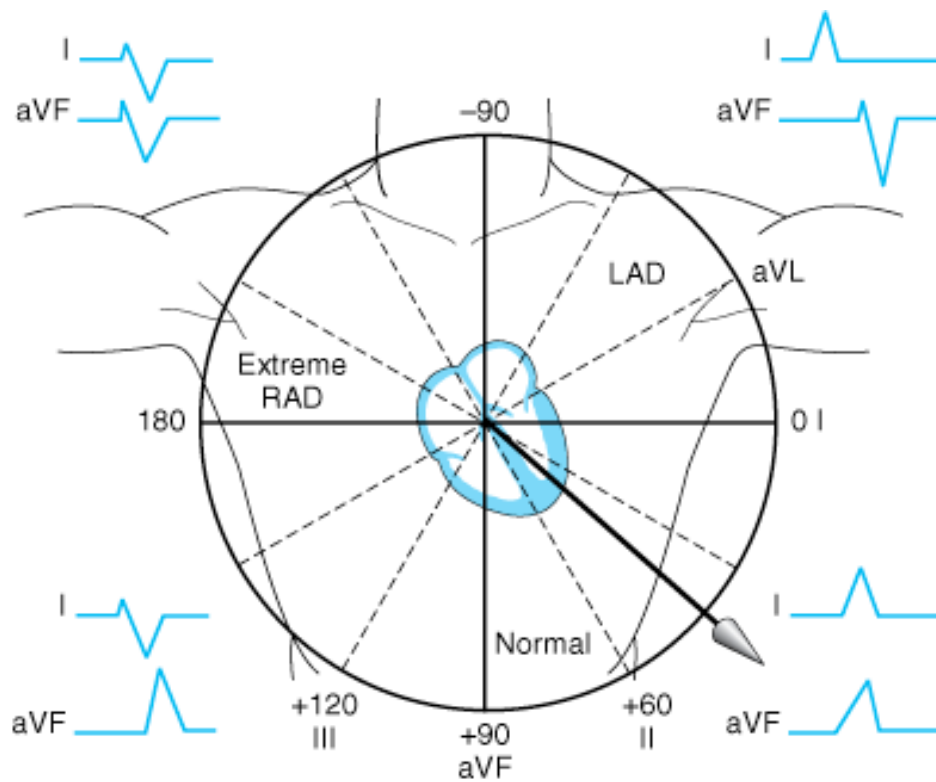
ravi.ranjan@hsc.utah.edu

Basics of ECG

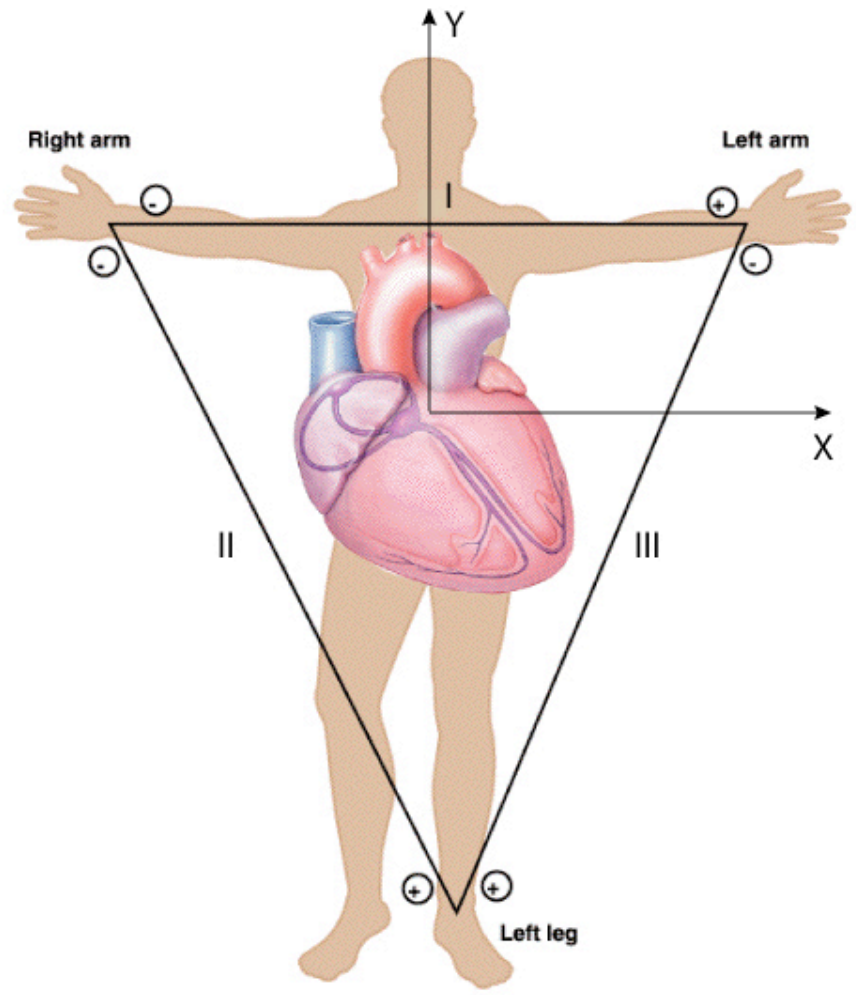
Normal Sinus Rhythm



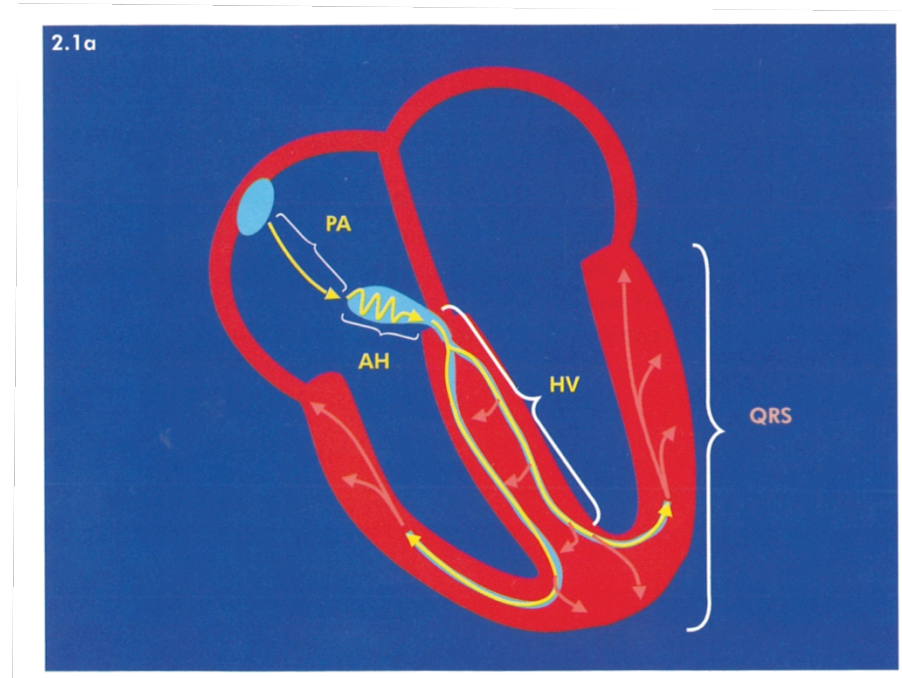
Normal duration of PR interval is 0.12-0.20 s (three to five small squares)



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Propagation in Normal Rhythm



Normal ECG

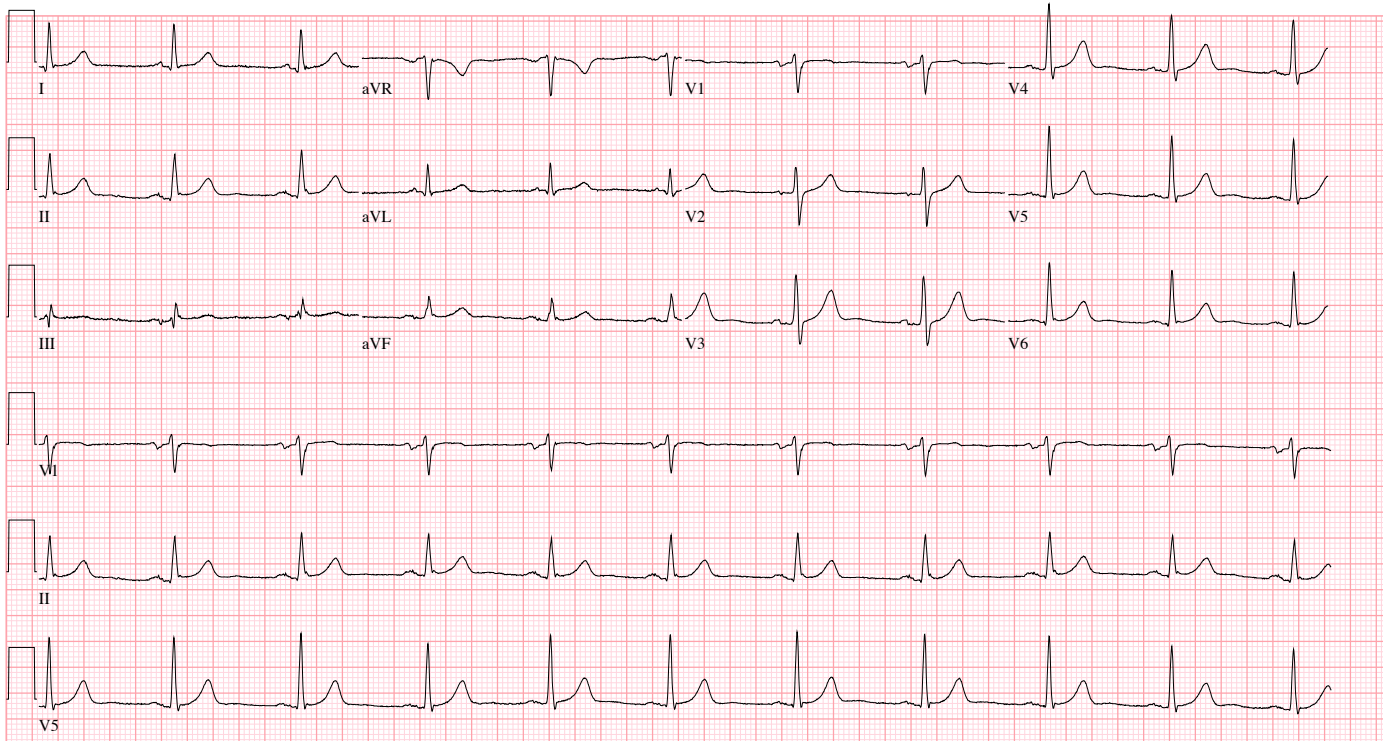
UNIVERSITY HEALTH CARE

Vent. rate	62	BPM
PR interval	154	ms
QRS duration	92	ms
QT/QTc	402/408	ms
P-R-T axes	36 38	40

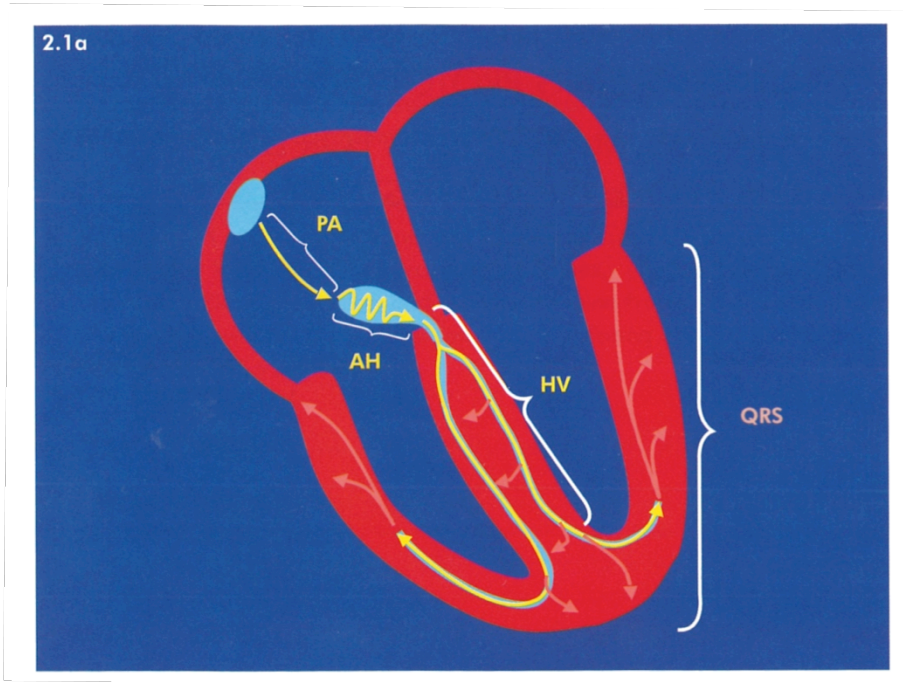
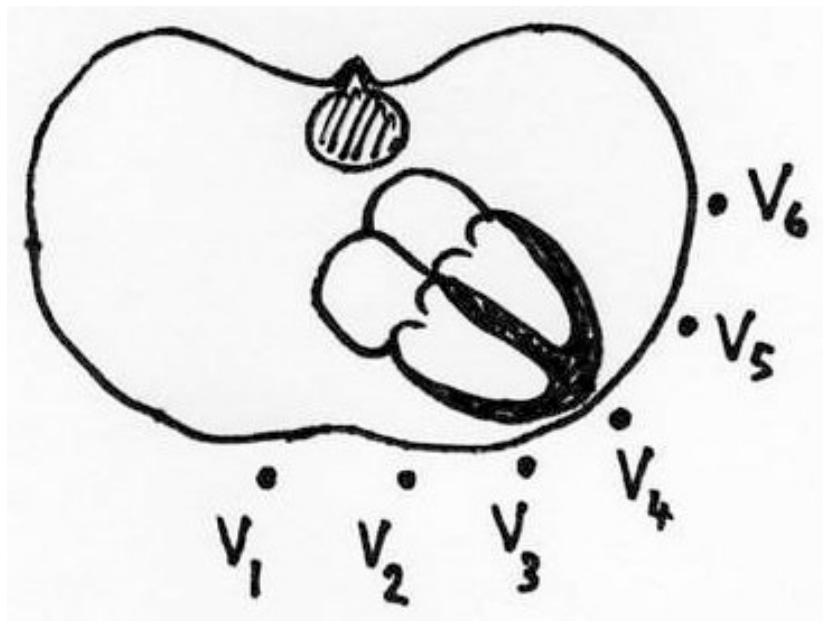
Technician: AMBER
Test ind:780.2

Referred by: MOHAMED HAMDAN

Confirmed By: Ravi Ranjan



Bundle Branch Block



RBBB

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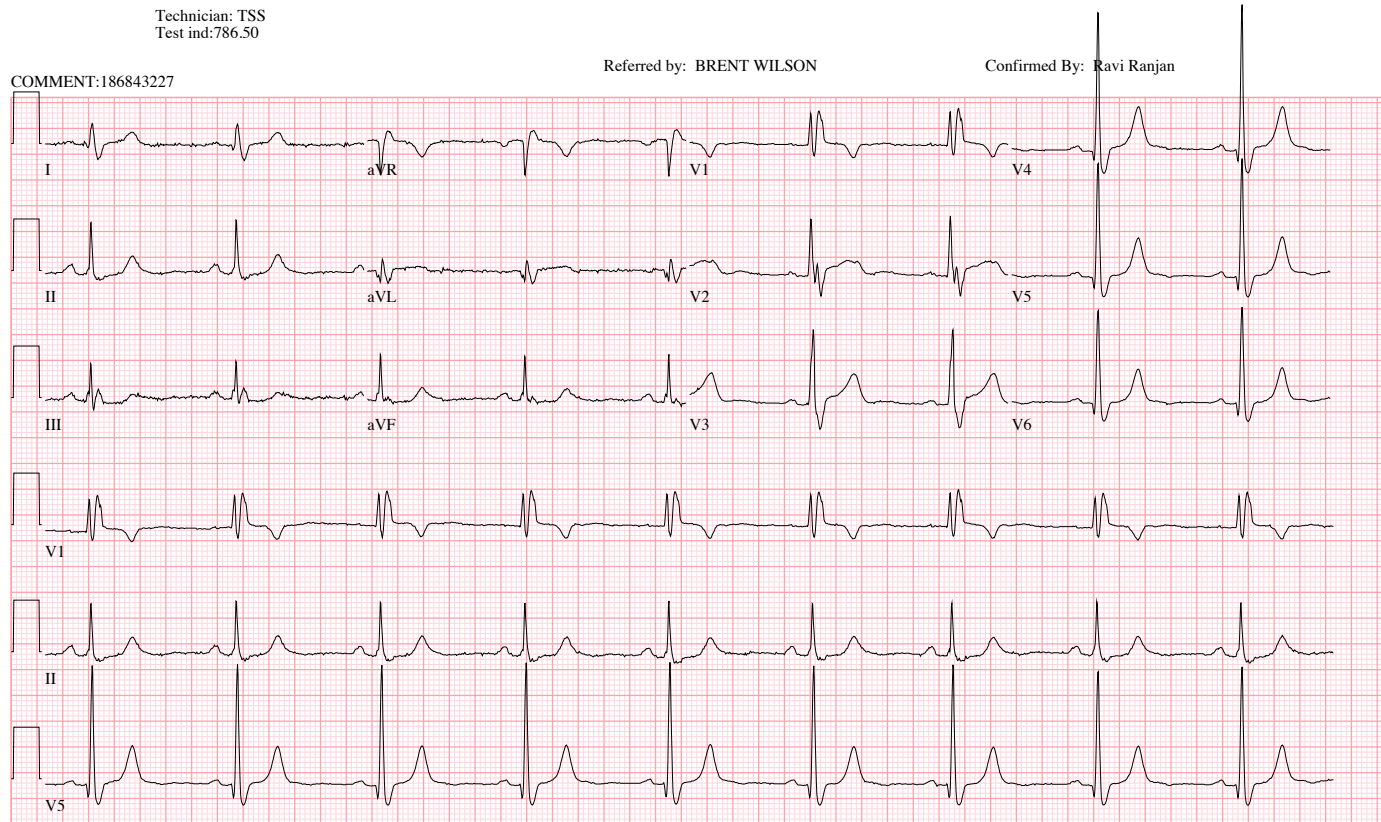
Vent. rate	53	BPM
PR interval	174	ms
QRS duration	138	ms
QT/QTc	458/429	ms
P-R-T axes	76 78	46

Technician: TSS
Test ind:786.50

Referred by: BRENT WILSON

Confirmed By: Ravi Ranjan

COMMENT:186843227



LBBB

UNIVERSITY HEALTH CARE

Vent. rate	64	BPM
PR interval	234	ms
QRS duration	184	ms
QT/QTc	516/532	ms
P-R-T axes	-23 -57	117

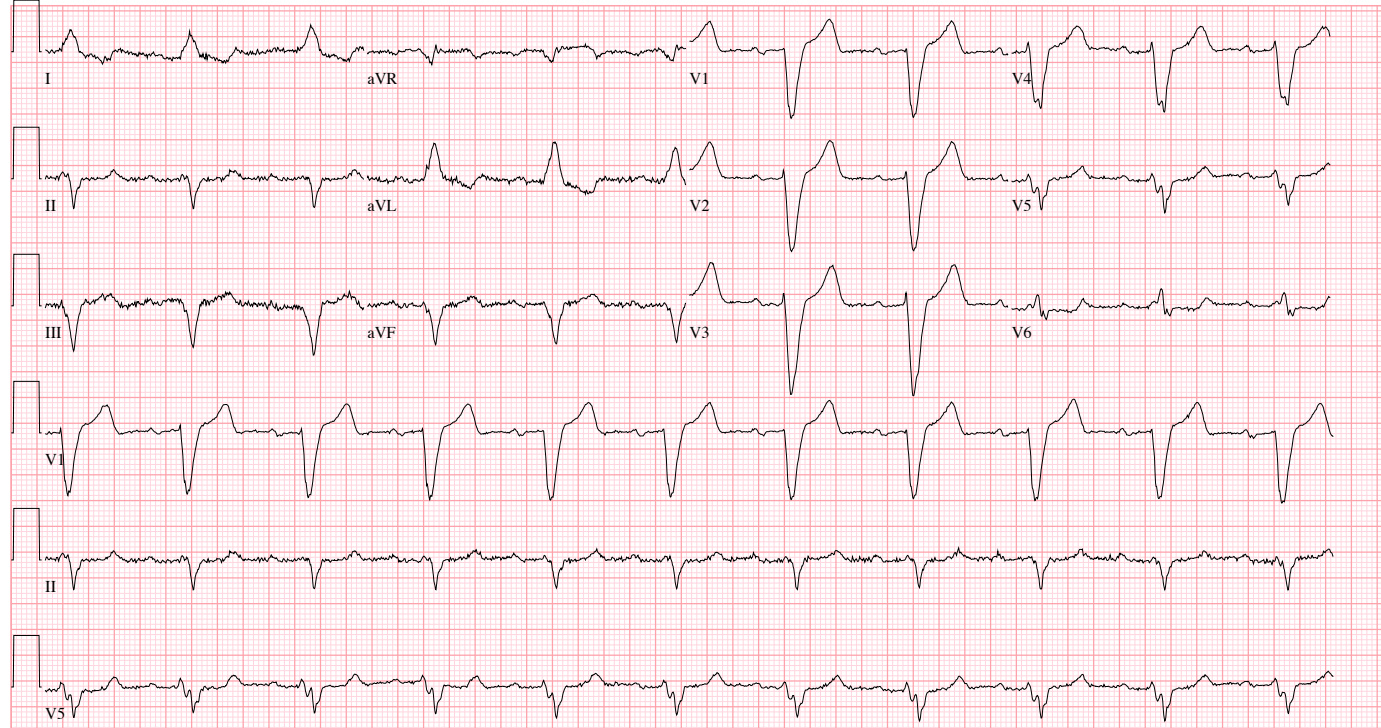
Technician: TSS
Test ind: VT

Referred by: HAMDAN

Confirmed By: Ravi Ranjan

Comment: 185310459

Study:



Sinus Bradycardia

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Vent. rate	46	BPM
PR interval	178	ms
QRS duration	84	ms
QT/QTc	470/411	ms
P-R-T axes	25 -2	29

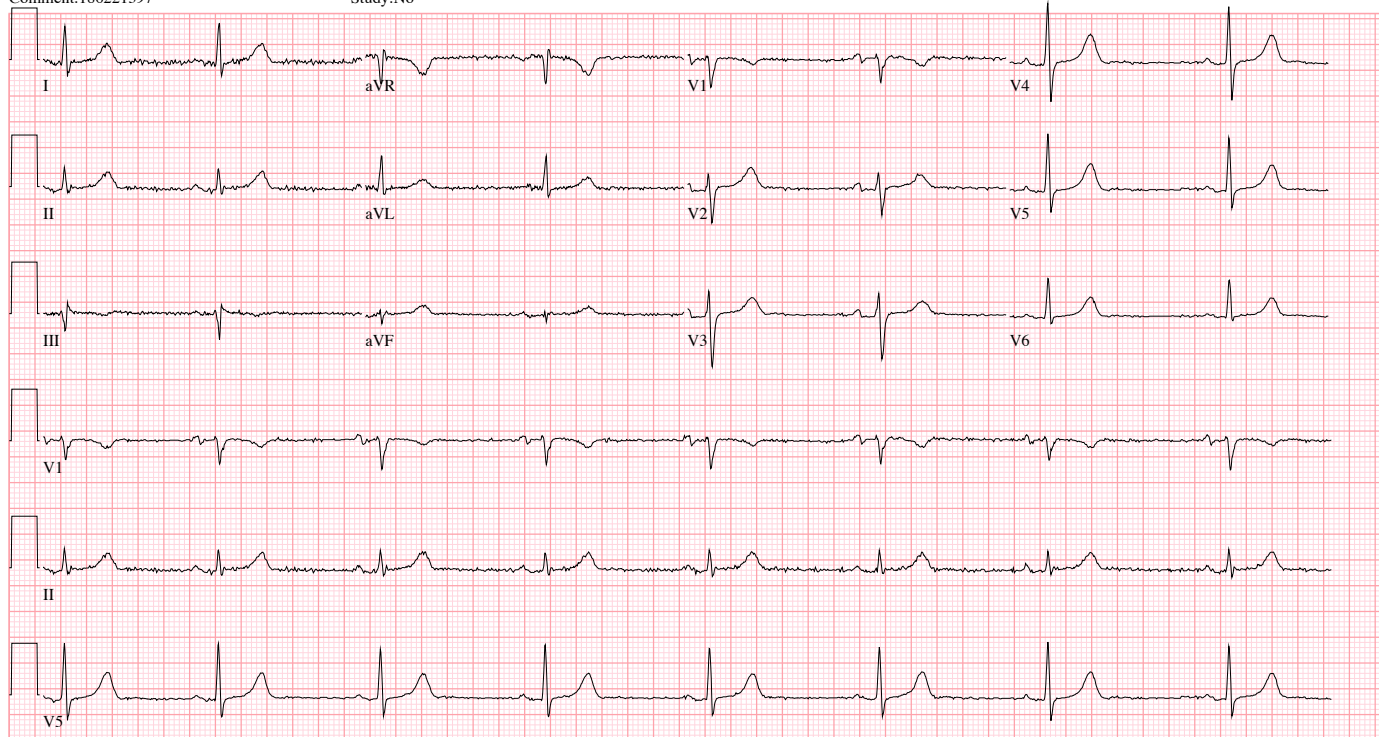
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Test ind: A FIB

Referred by: NASSIR MARROUCHE

Confirmed By: Ravi Ranjan

Comment: 186221397

Study: No



Sinus Tachycardia

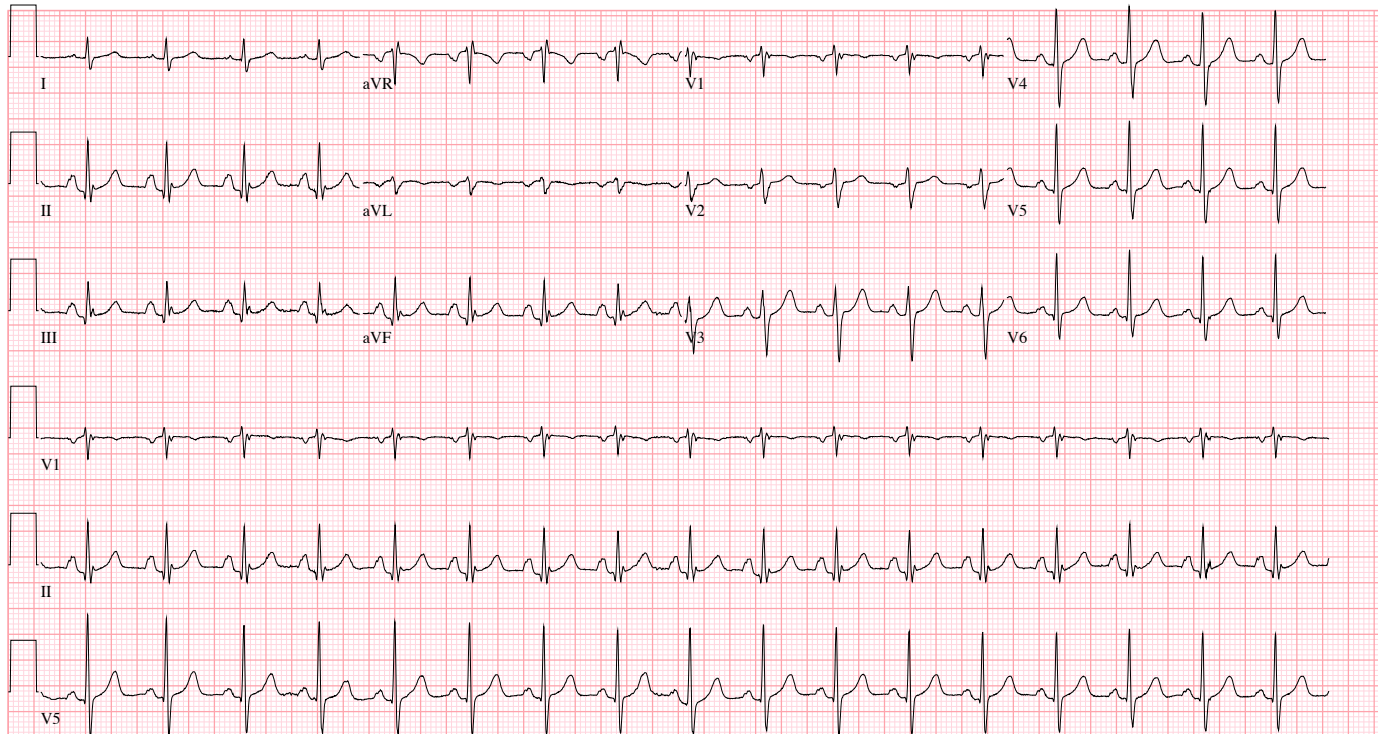
UNIVERSITY HEALTH CARE

Vent. rate	104	BPM
PR interval	132	ms
QRS duration	86	ms
QT/QTc	326/428	ms
P-R-T axes	80 75	72

Technician: AMBER
Test ind:780.2

Referred by: NATALIE SANDERS

Confirmed By: Ravi Ranjan



Atrial Flutter

ID:003932084

UNIVERSITY HEALTH CARE

Vent. rate	113	BPM
PR interval	*	ms
QRS duration	98	ms
QT/QTc	324/444	ms
P-R-T axes	84 81 76	

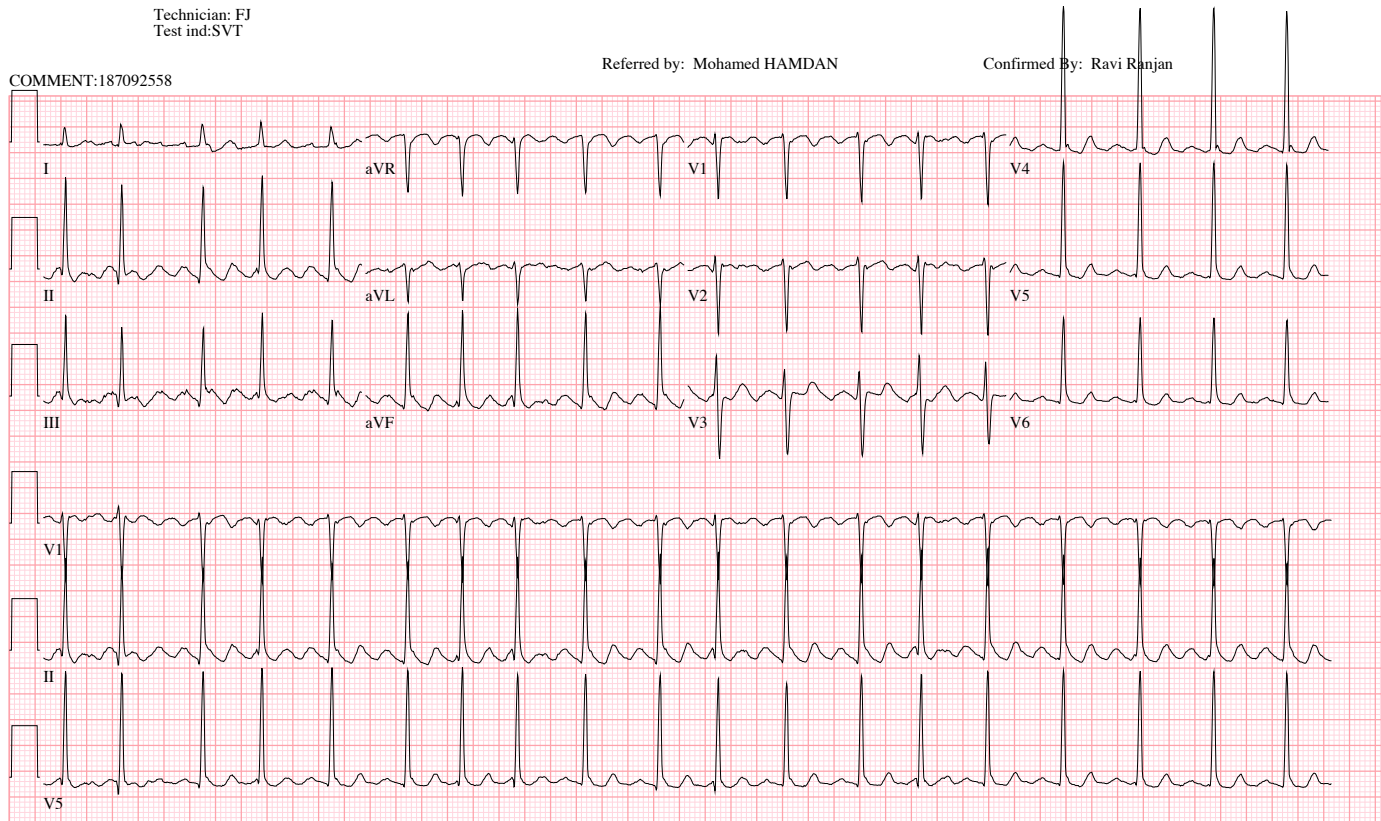
9:18,

Technician: FJ
Test ind:SVT

Referred by: Mohamed HAMDAN

Confirmed By: Ravi Ranjan

COMMENT:187092558



Atrial Fibrillation

ID:010127587

UNIVERSITY HEALTH CARE

Vent. rate	72	BPM
PR interval	*	ms
QRS duration	62	ms
QT/QTc	406/444	ms
P-R-T axes	* 26	70

4:09.

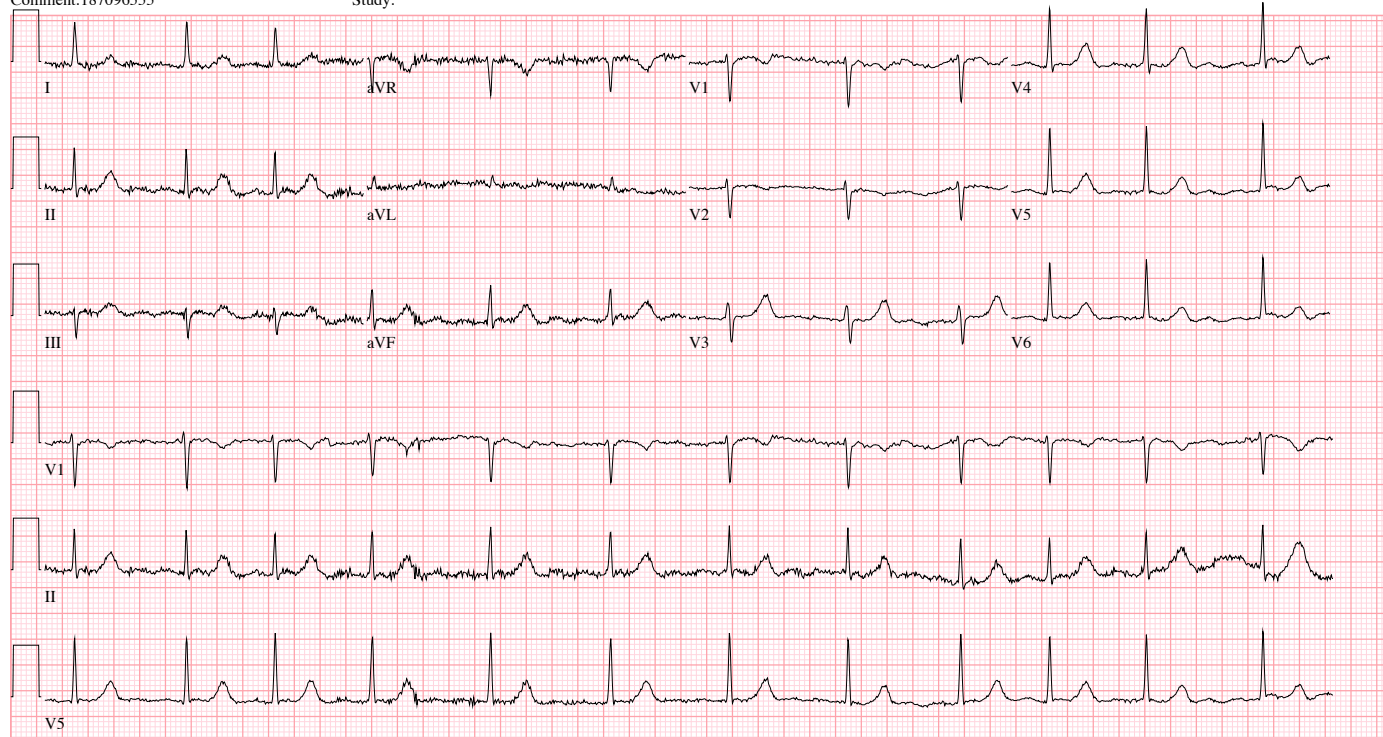
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Referred by: CRAIG SELZMAN

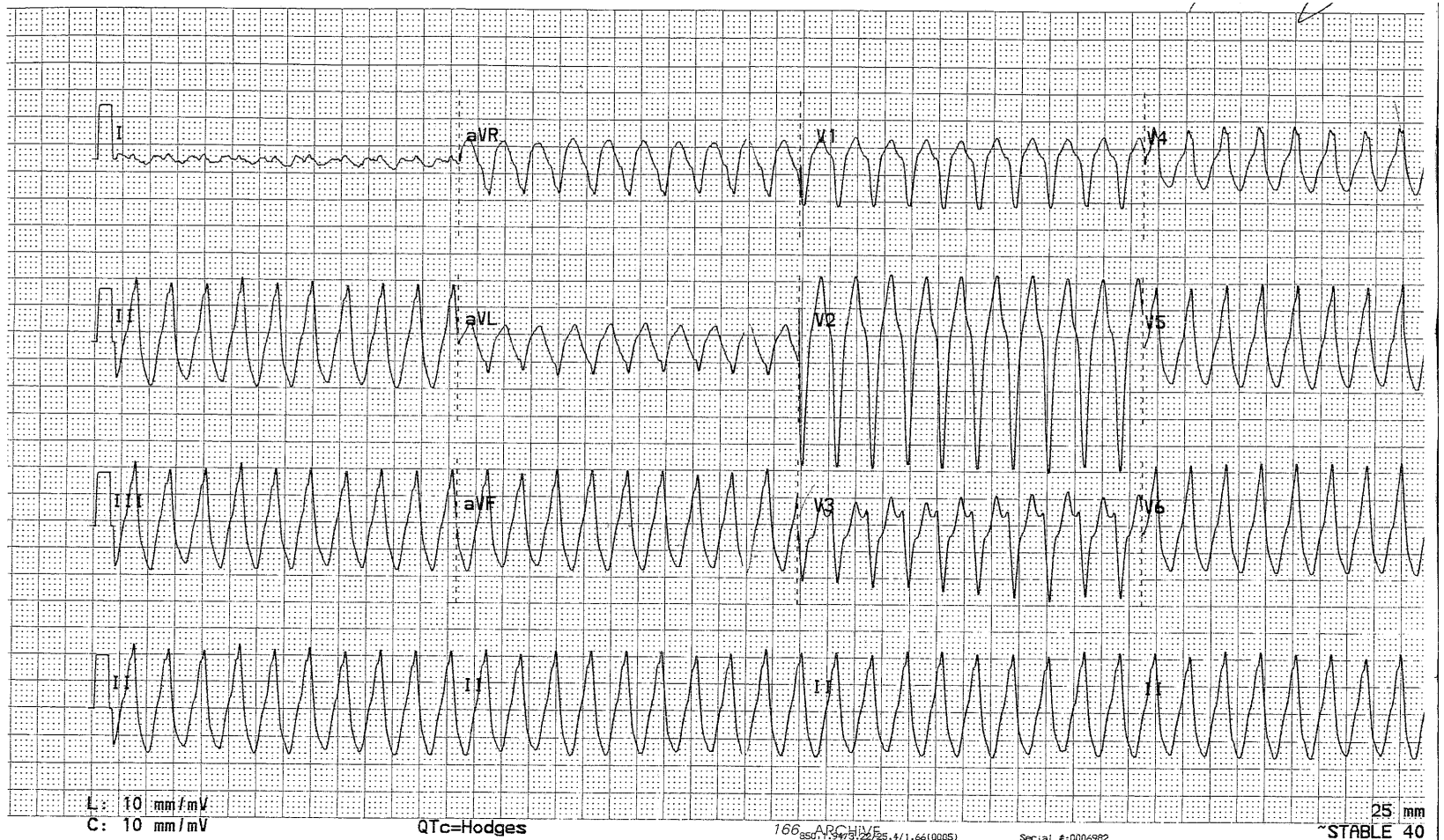
Confirmed By: Ravi Ranjan

Comment: 187096555

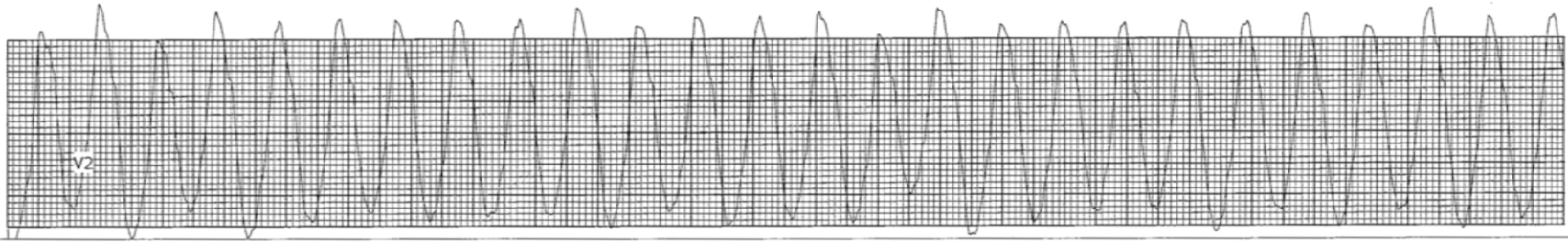
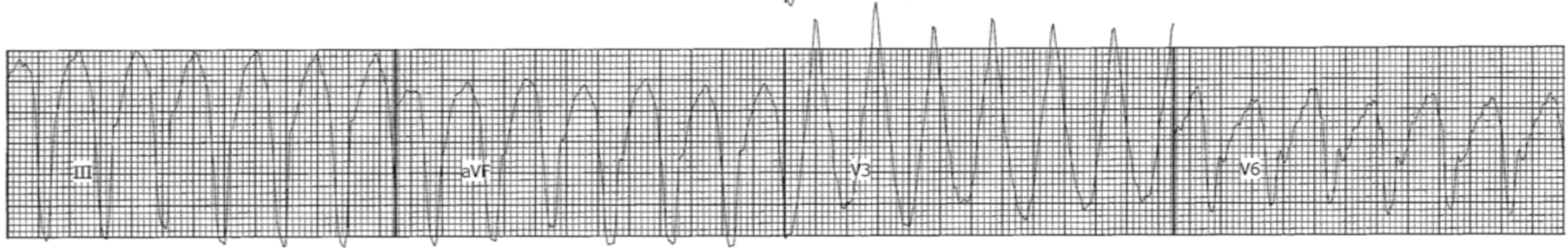
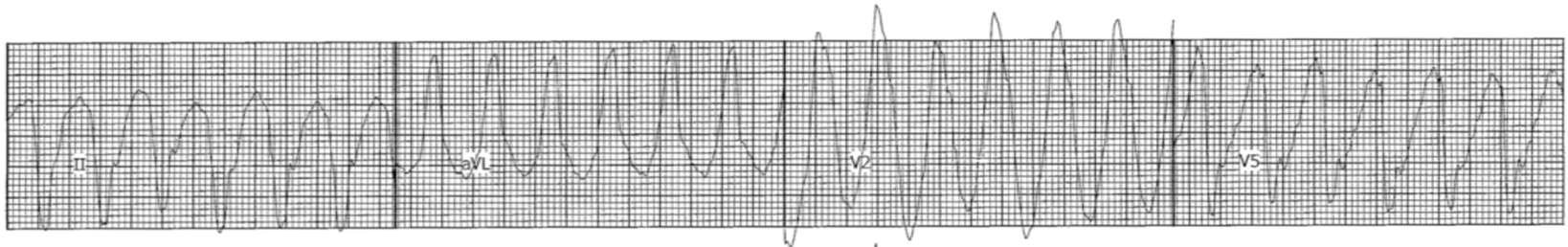
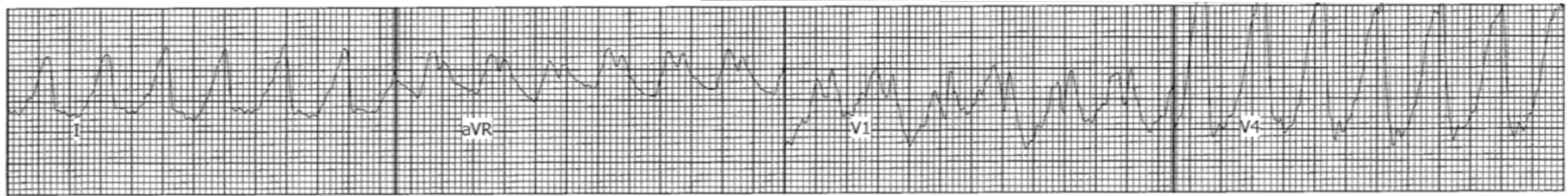
Study:



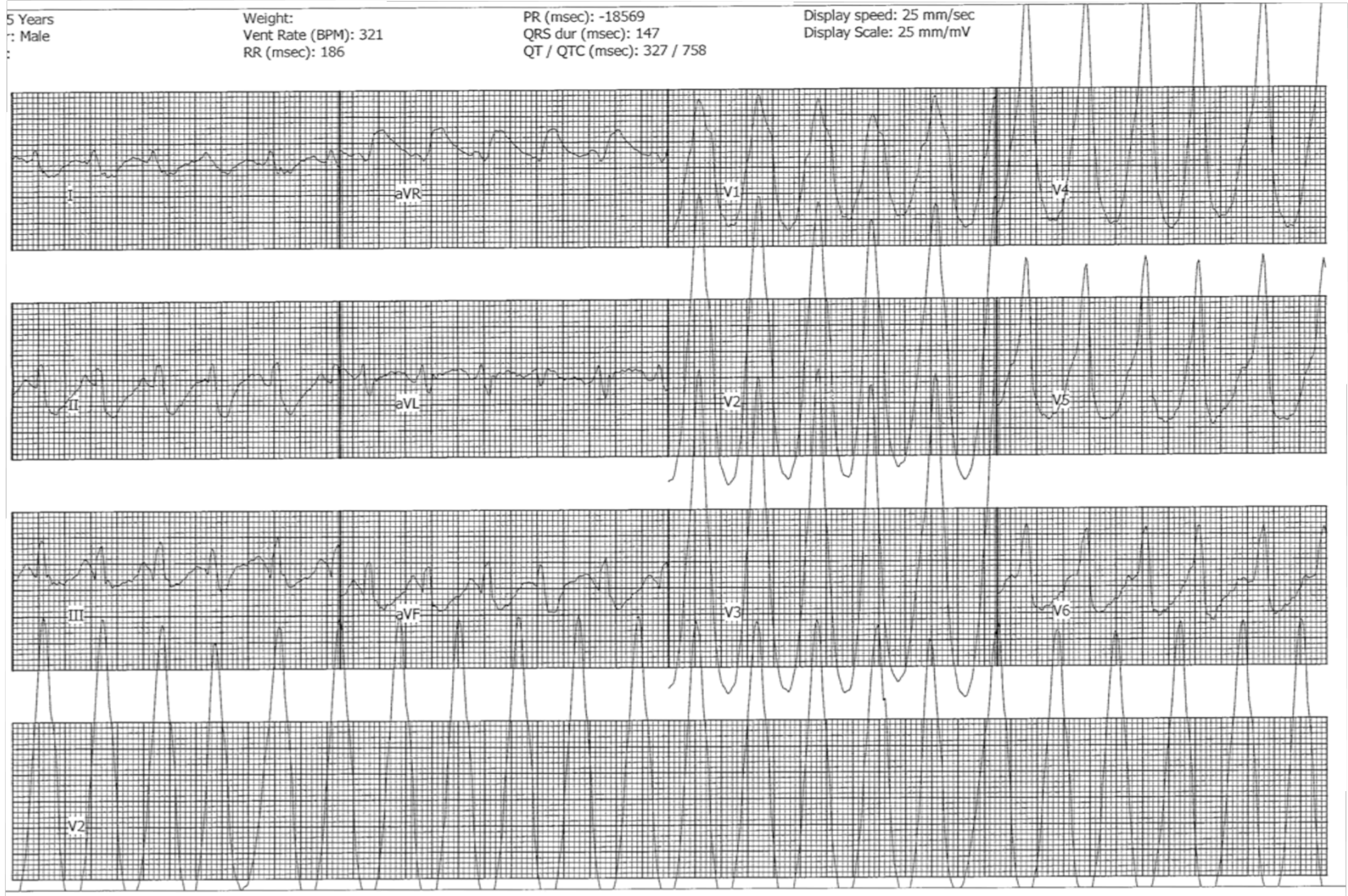
Ventricular Tachycardia



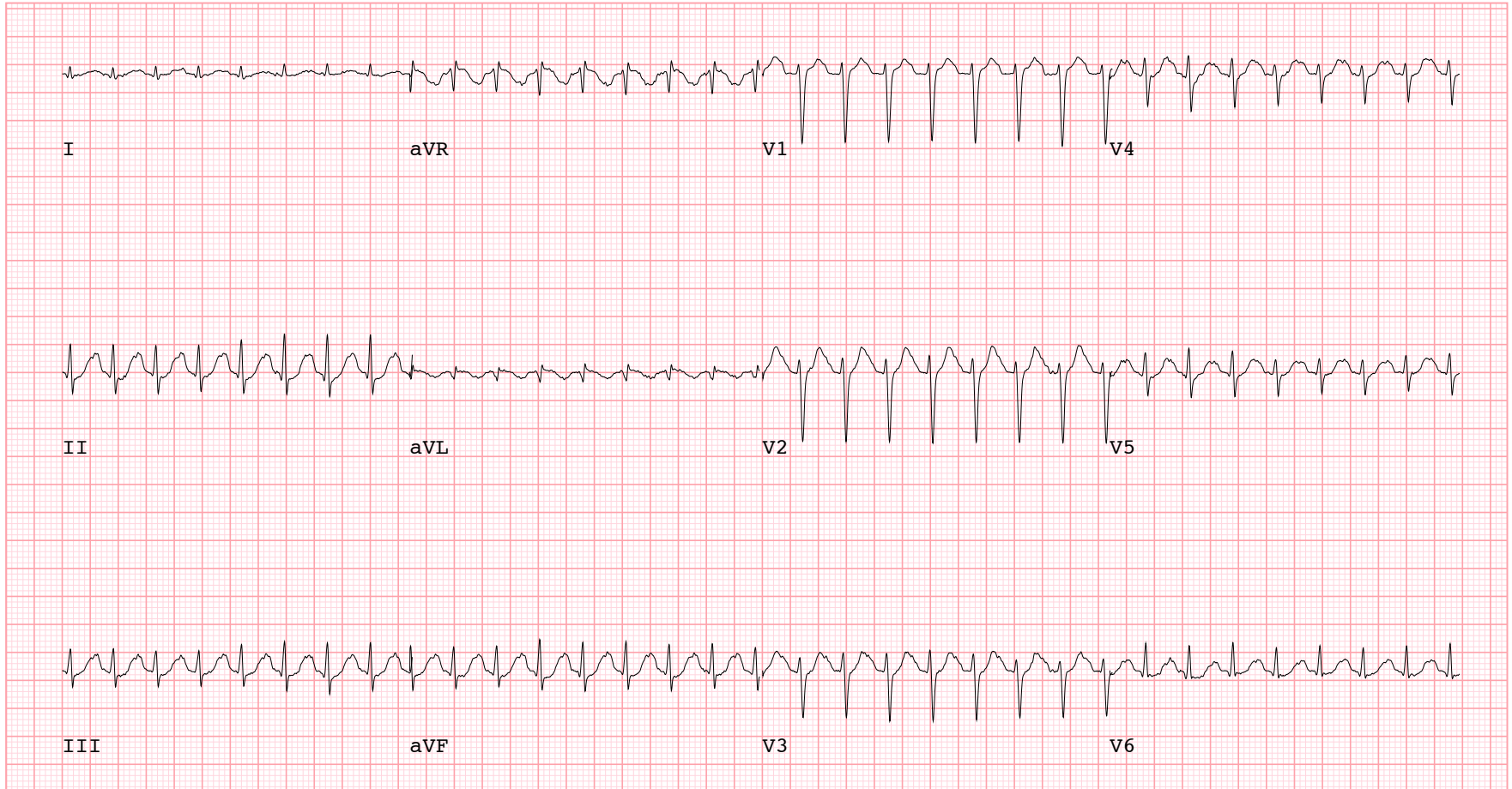
Ventricular Tachycardia



Ventricular Tachycardia



Supra Ventricular Tachycardia



GE Medical Systems IT
CASE V5.02

25mm/s 10mm/mV 60Hz 0.01-40Hz S+ HR(V1,V2)

Basic EP recordings

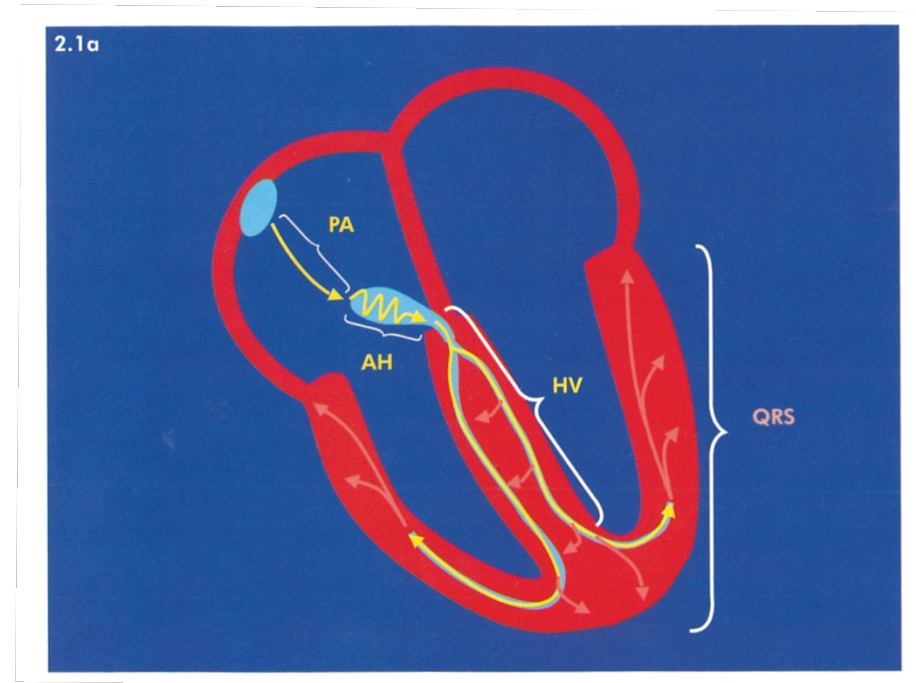
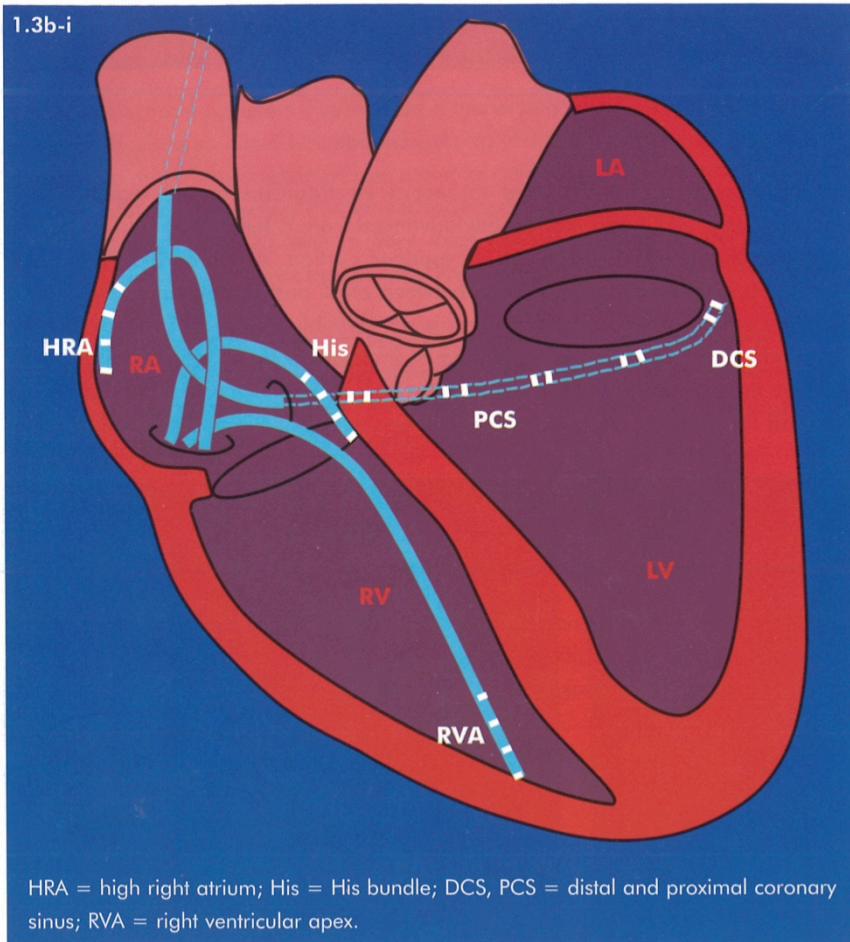


Figure 1.3b-i illustrates the standard catheter positions for a 'four-wire' diagnostic EP study.

Basic EP recordings

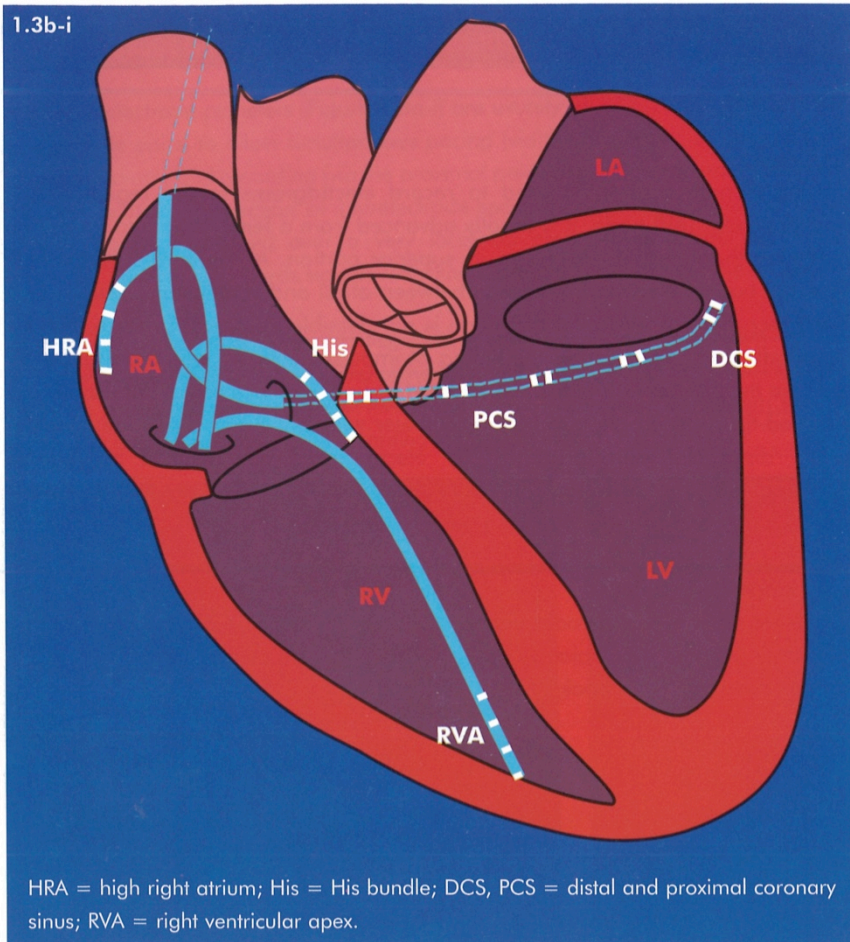


Figure 1.3b-i illustrates the standard catheter positions for a 'four-wire' diagnostic EP study.

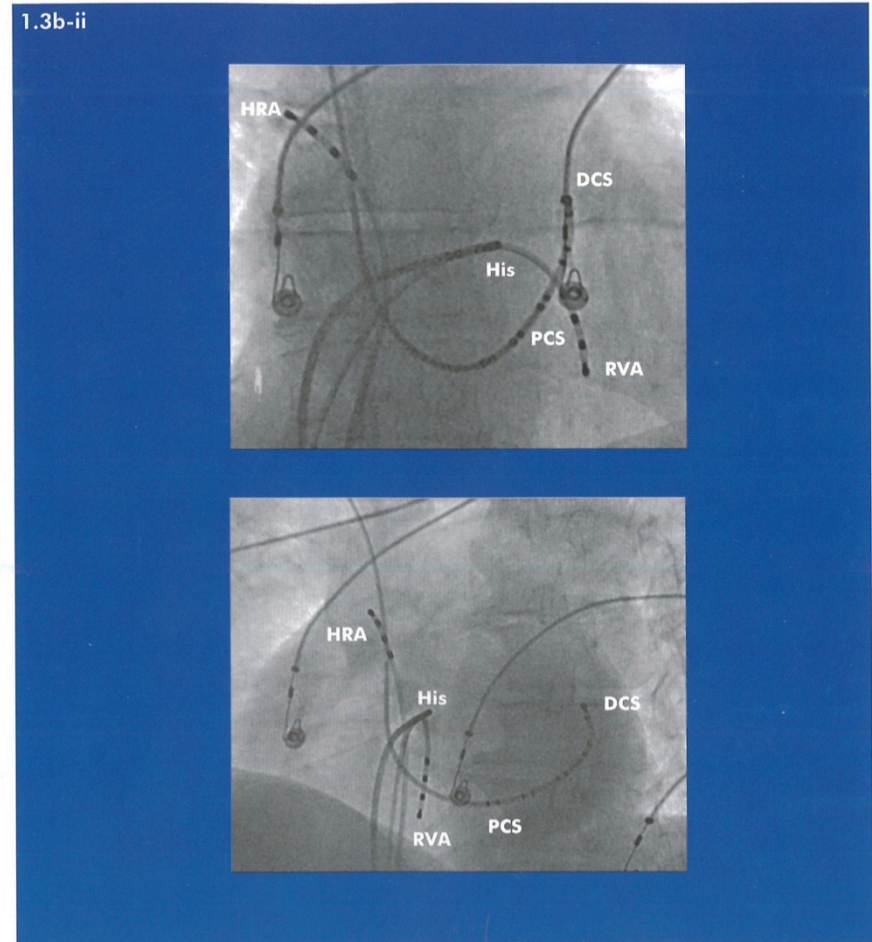


Figure 1.3b-ii shows the catheter positions for a standard 'four-wire' diagnostic EP study in the posteroanterior (top) and left anterior oblique (bottom) fluoroscopic projections. Abbreviations as in Figure 1.3b-i.

Basic EP recordings



Tracing 1.3 Electrograms displayed during standard four-wire study in sinus rhythm. Although all twelve surface ECG leads are recorded, only three approximately orthogonal leads are shown, for clarity. The right ventricular apex (RV) and high right atrium (HRA) leads show sharp single chamber electrograms. The His bundle catheter records activity adjacent to the AV node; the distal bipole (HBE D) favoring the His bundle electrogram (H) and the adjacent ventricular myocardium (V), while the proximal bipole (HBE P) shows a large atrial electrogram (A). Note that, although the ventricular spike recorded by the His bundle comes from tissue adjacent to the bundle of His, the earliest ventricular activity is at the apex (RV). The electrograms recorded by the bipoles of the decapolar coronary sinus catheter are labeled CS 9–10 (proximal) to CS 1–2 (distal); each shows a sharp atrial electrogram followed by a smaller ventricular electrogram.

WPW

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Vent. rate	56	BPM
PR interval	80	ms
QRS duration	164	ms
QT/QTc	470/453	ms
P-R-T axes	106 41	45

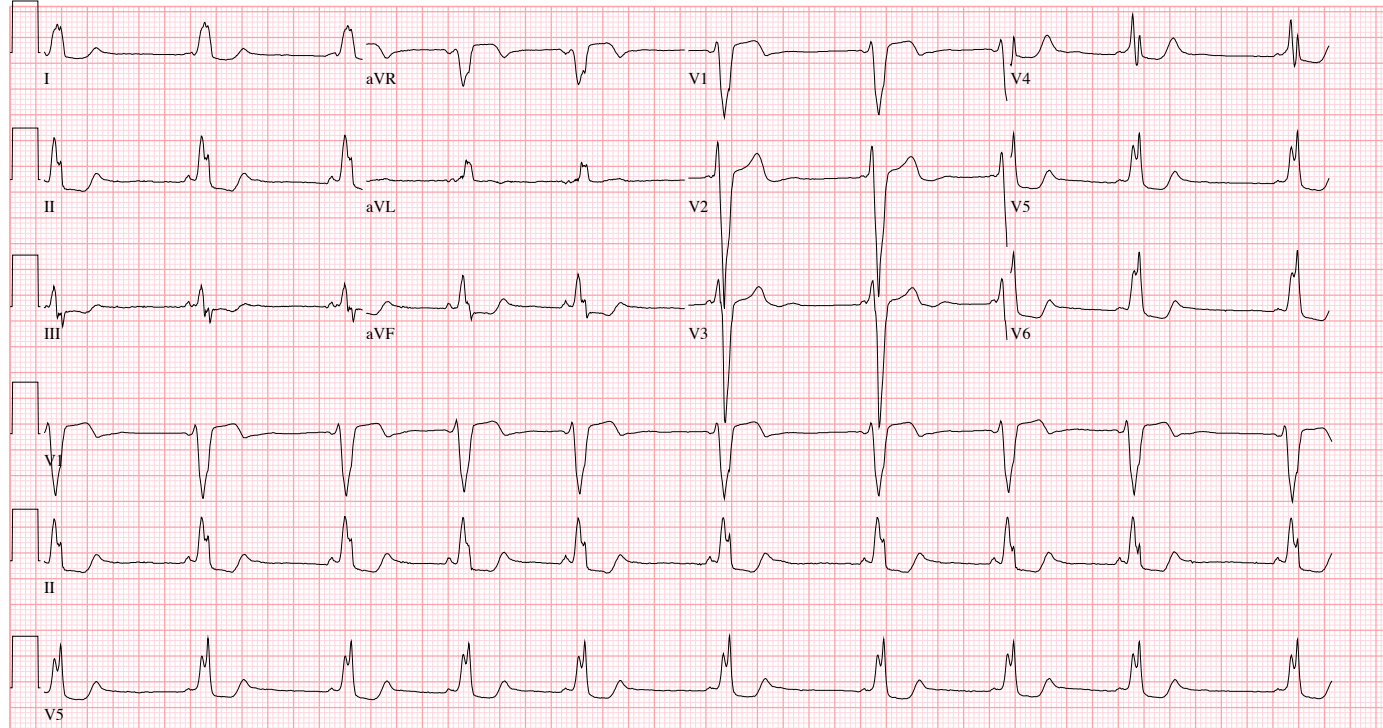
Technician: TSS
Test ind:426.7

Referred by: Roger FREEDMAN

Confirmed By: Mohamed HAMDAN

Comment:186170738

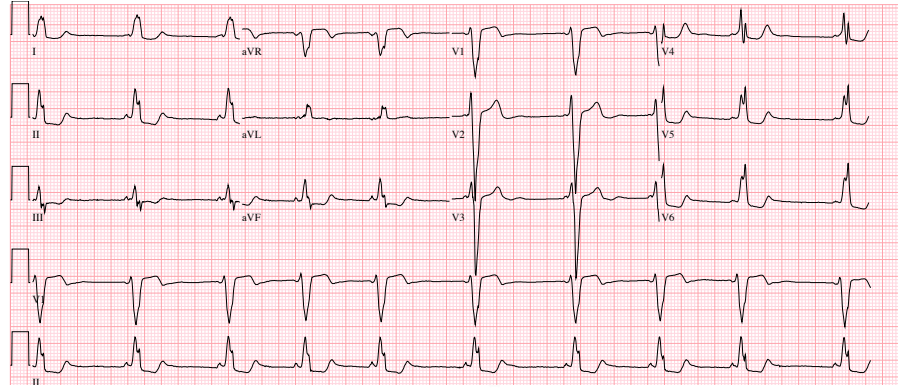
Study:



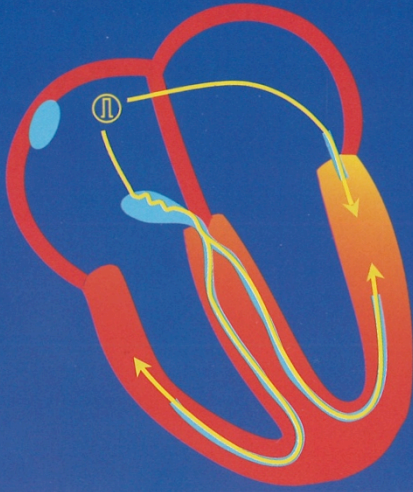
25mm/s 10mm/mV 150Hz 7.1.1 12SL 239 CID: 70

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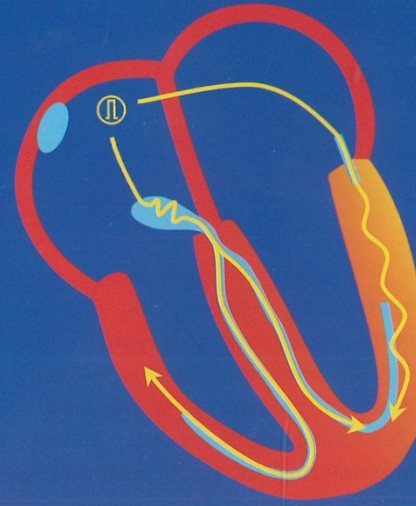
WPW



5.1a



5.1b

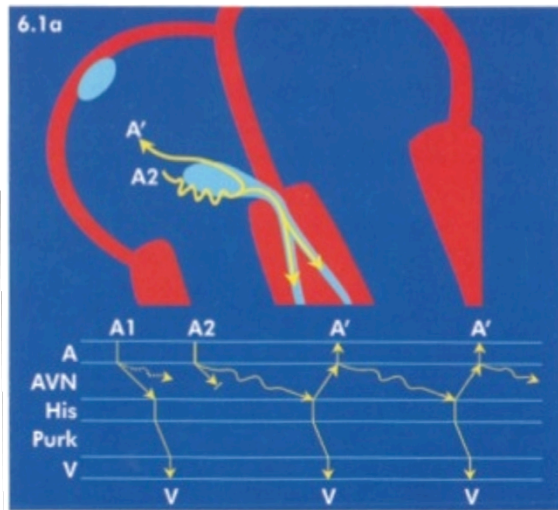


Supra Ventricular Tachycardia (SVT)

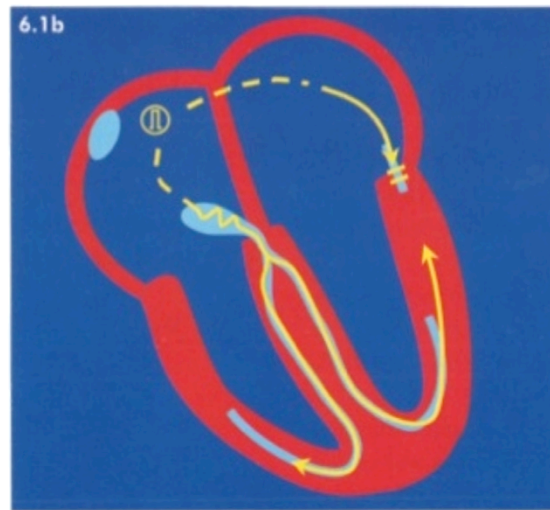
- AVNRT – AV nodal re-entrant tachycardia
- AVRT – Atrio-ventricular re-entrant tachycardia
- AT – atrial tachycardia

SVT initiation

AVNRT
AV nodal re-entrant
tachycardia



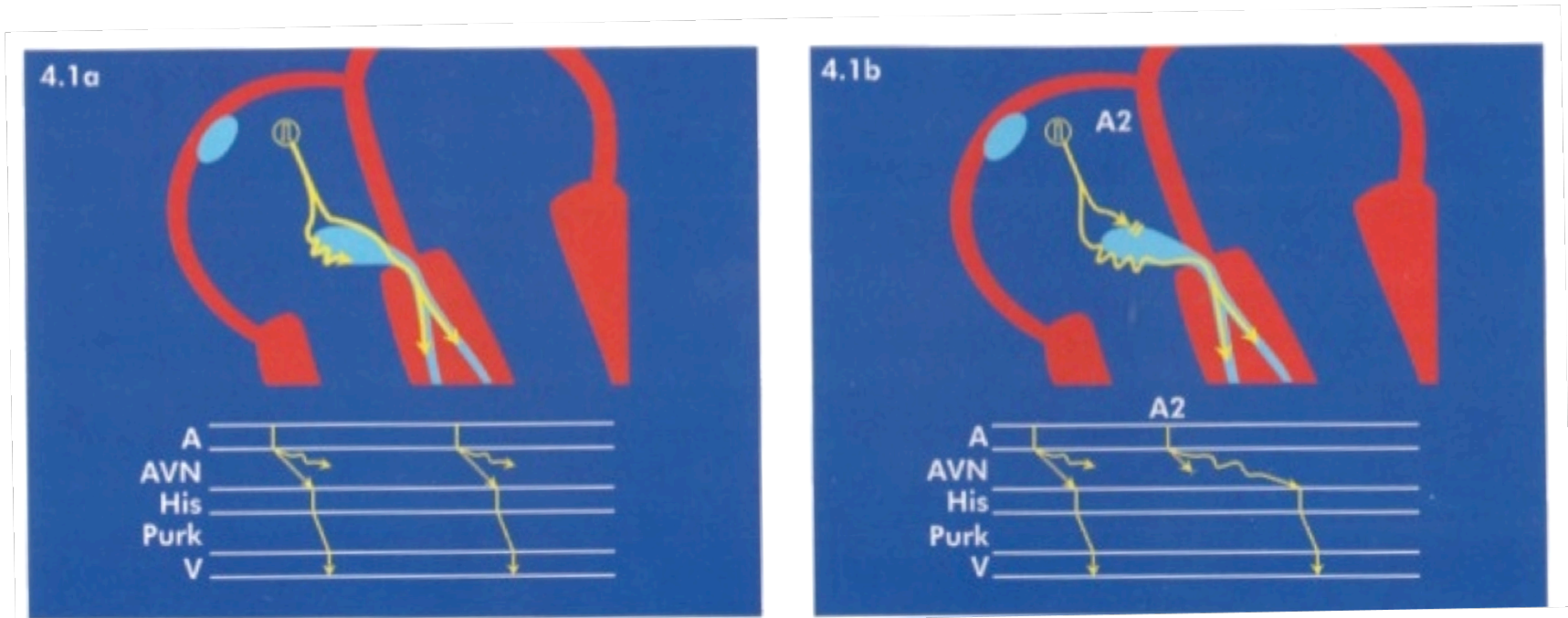
ORT
Orthodromic re-entrant
tachycardia



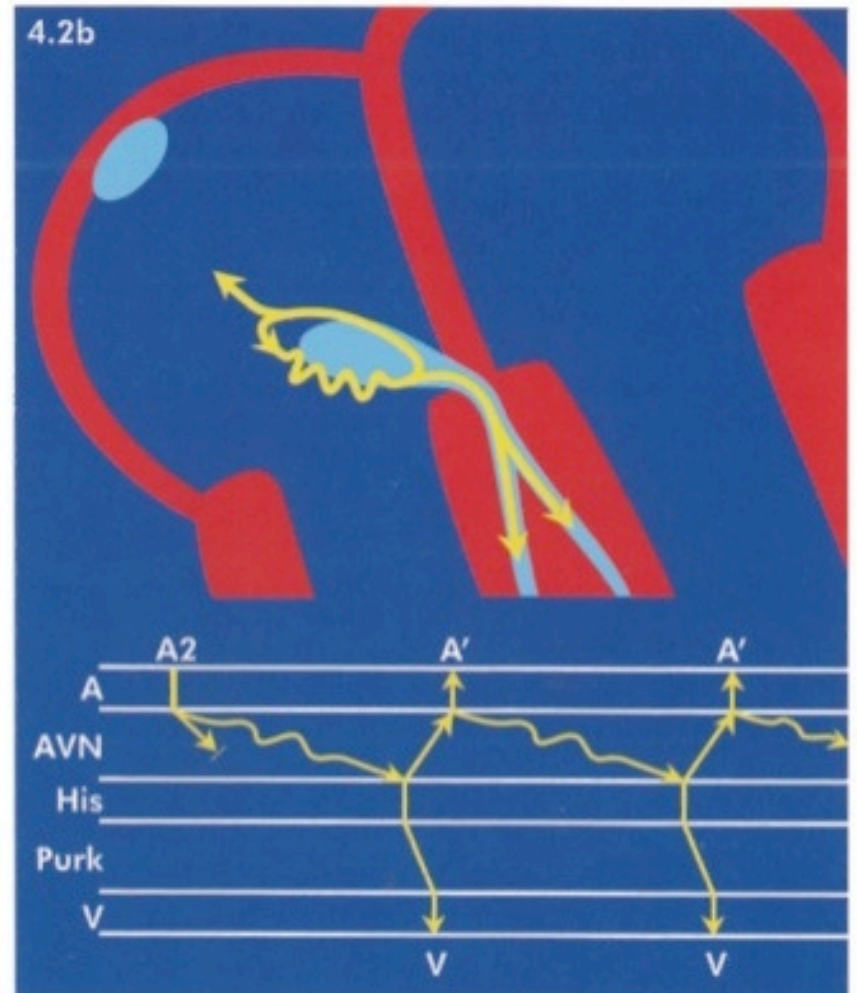
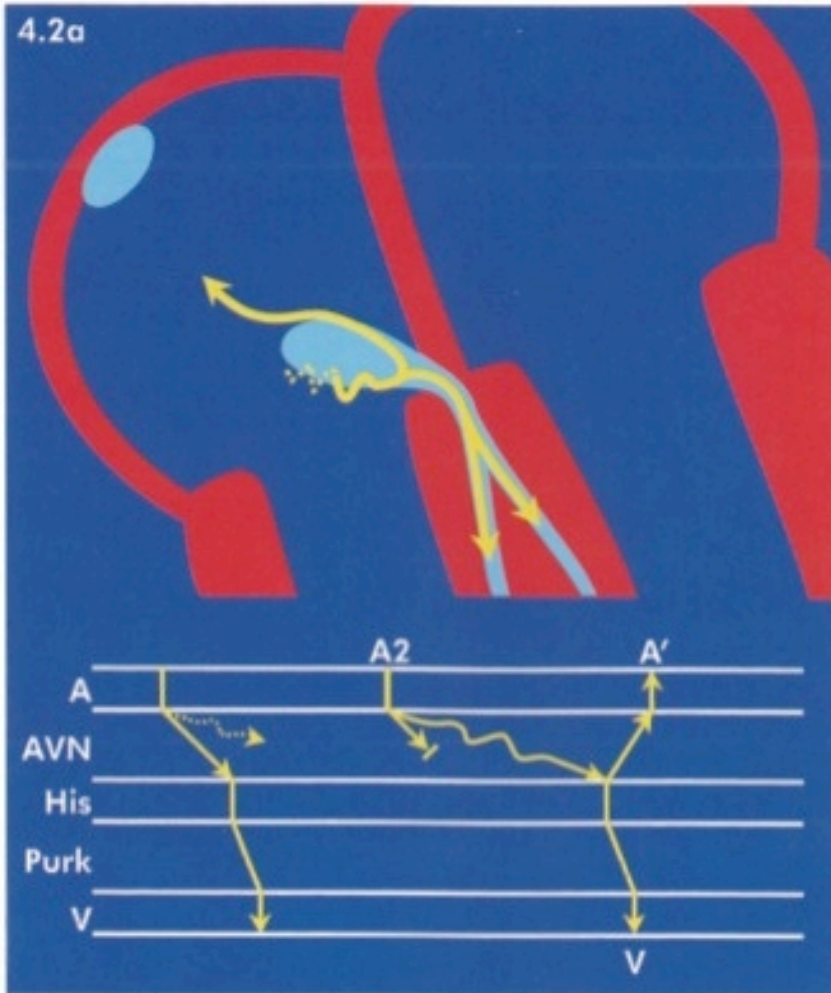
AT
Atrial Tachycardia



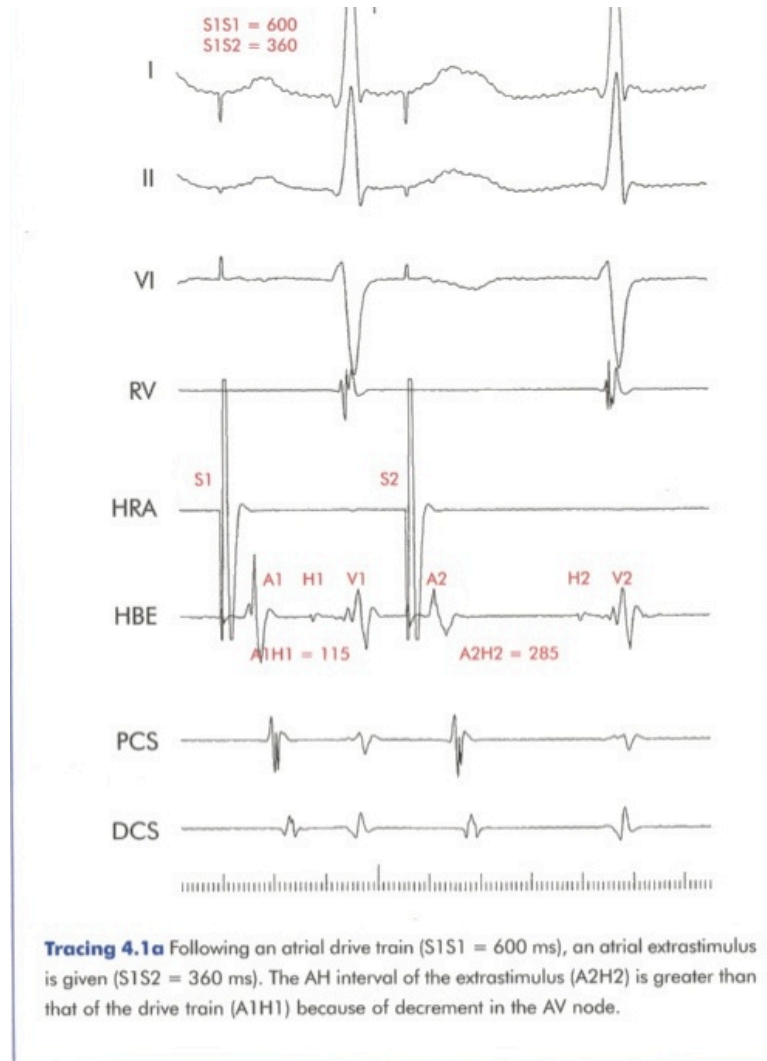
Dual Node Pathway



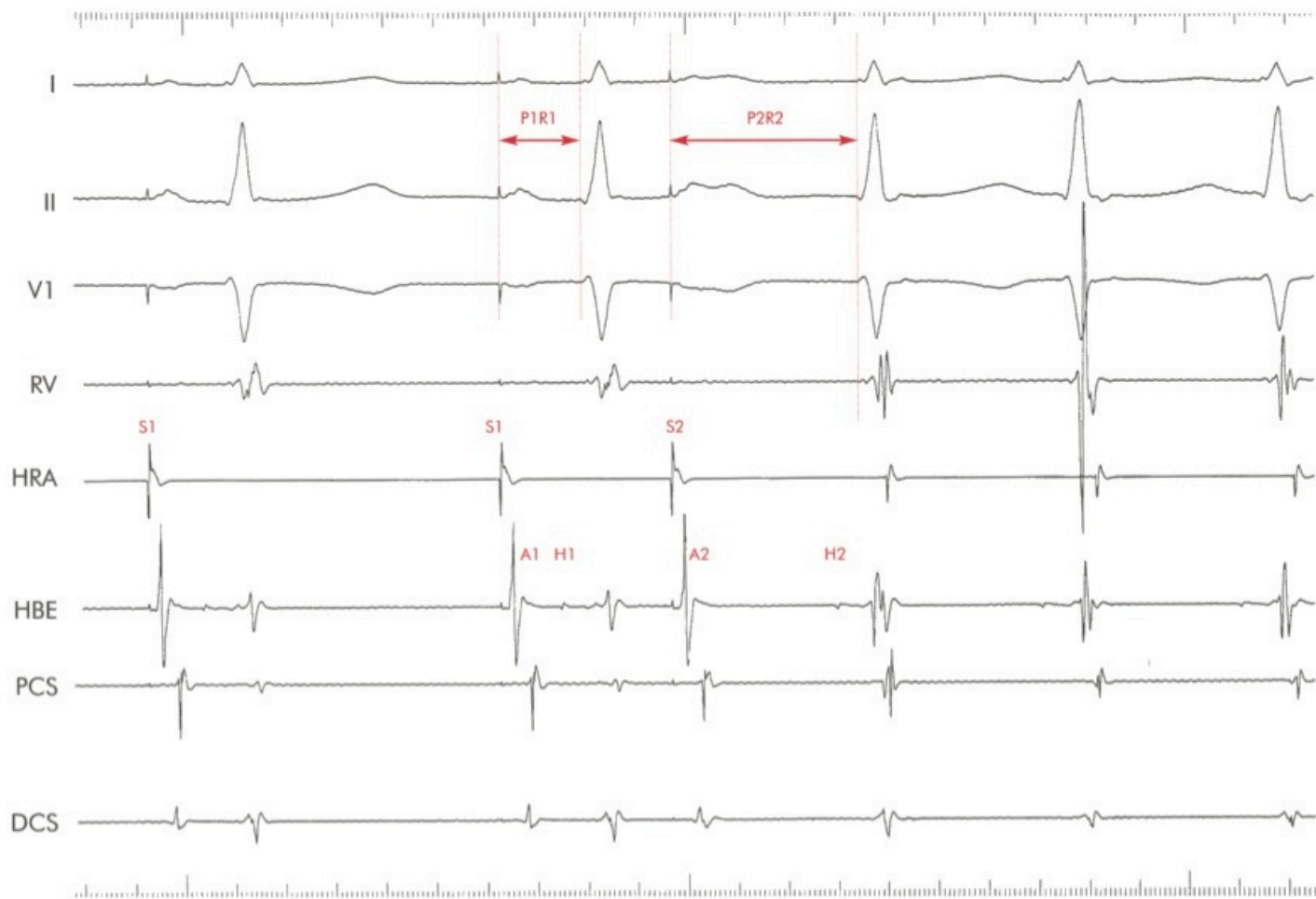
AVNRT



Dual Node Pathway



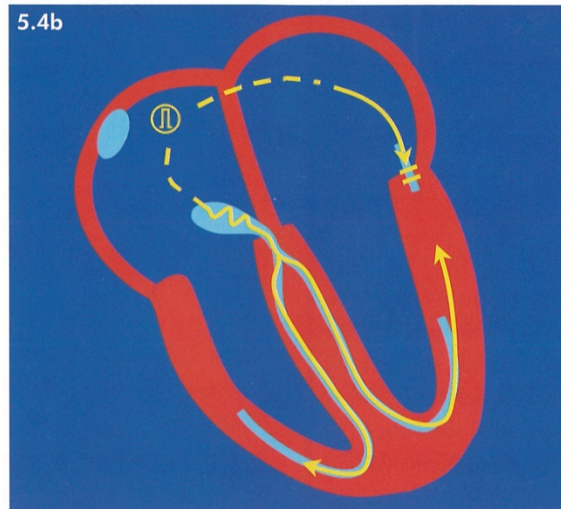
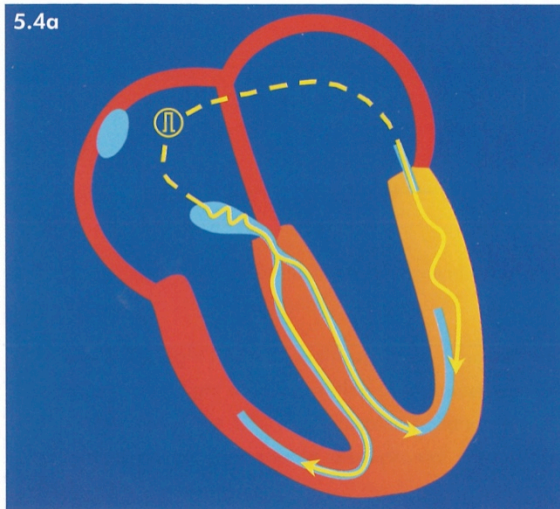
AVNRT



Tracing 6.1a The onset of tachycardia may differ according to the tachycardia mechanism. AV nodal reentry is induced by an atrial extrastimulus, which results in block in the fast pathway and conduction over the slow pathway. The AH prolongation is obvious but the mechanism can also be seen in the surface leads.

AVRT – initiation

Orthodromic Re-entrant Tachycardia (ORT)



ORT



Tracing 6.1b Orthodromic AVRT is induced by an atrial extrastimulus. The key element of induction of tachycardia is block in the accessory pathway with loss of pre-excitation (*), allowing retrograde conduction up the accessory pathway to the atrium, thereby completing the circuit (see Section 5.4).

AT



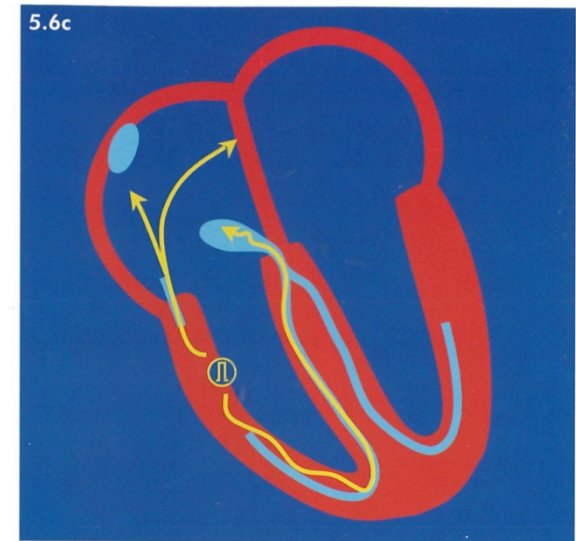
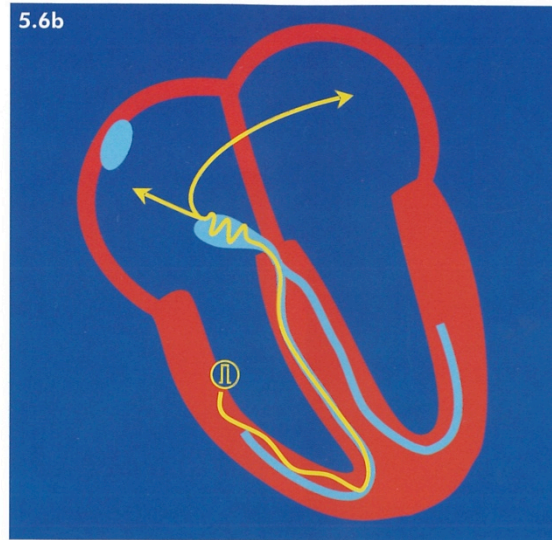
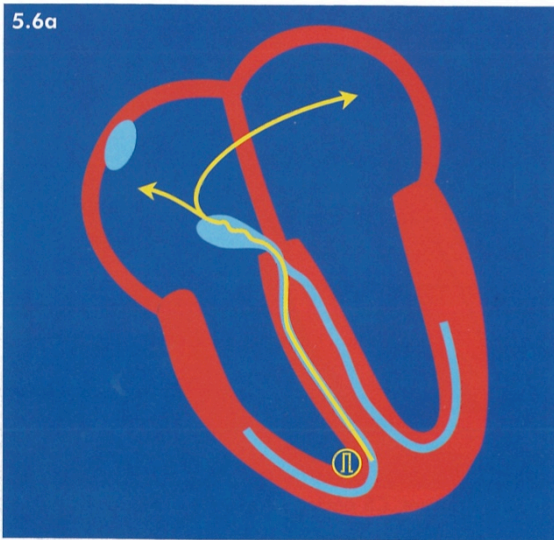
Tracing 6.1c In this example, the onset of atrial tachycardia is accompanied by a trivial increase in the AH interval, related to the increased atrial rate. However, subtle changes occur in the P-wave morphology between sinus rhythm (P) and tachycardia (P'), which correspond to slight changes in the atrial activation sequence seen on the intracardiac recordings. The observed AH interval makes AV reentry and AV node reentry very unlikely, since these are almost always associated with obvious AH prolongation.

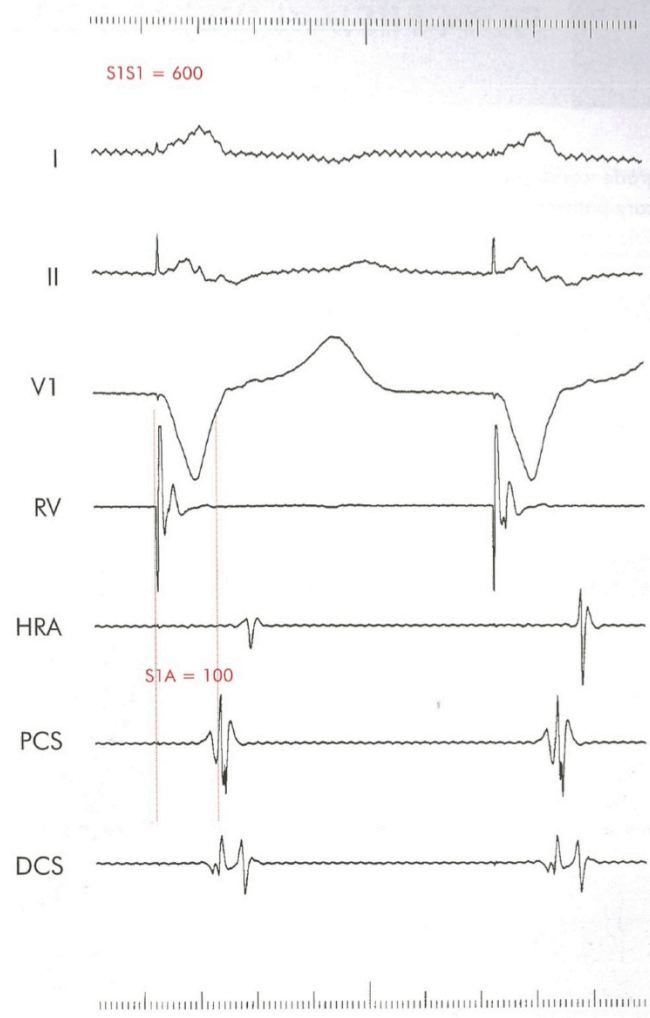
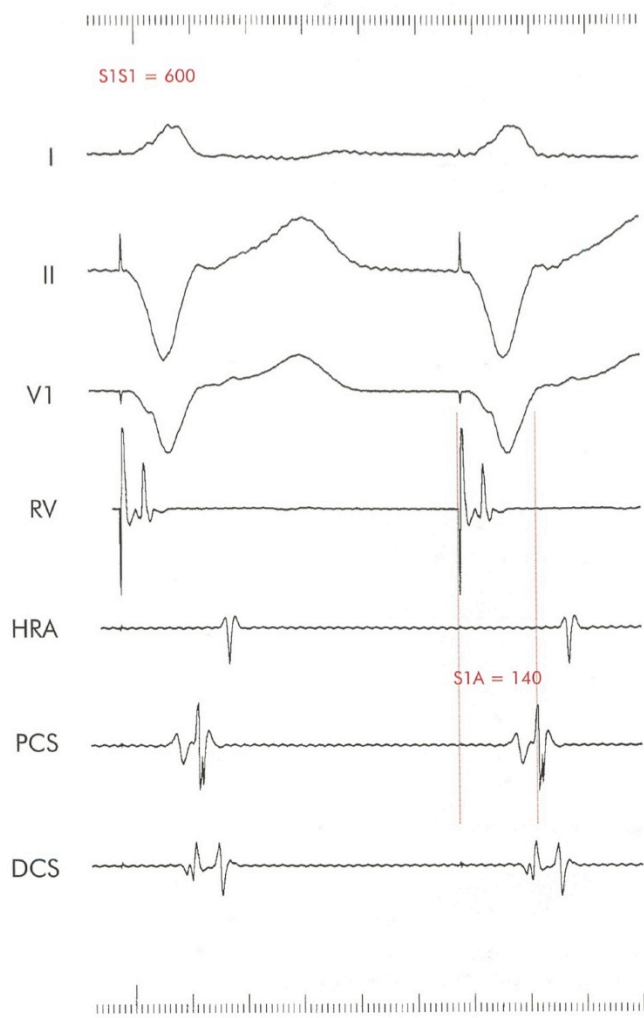
Base vs Apex Pacing

Apex Pacing

Base Pacing
No AP

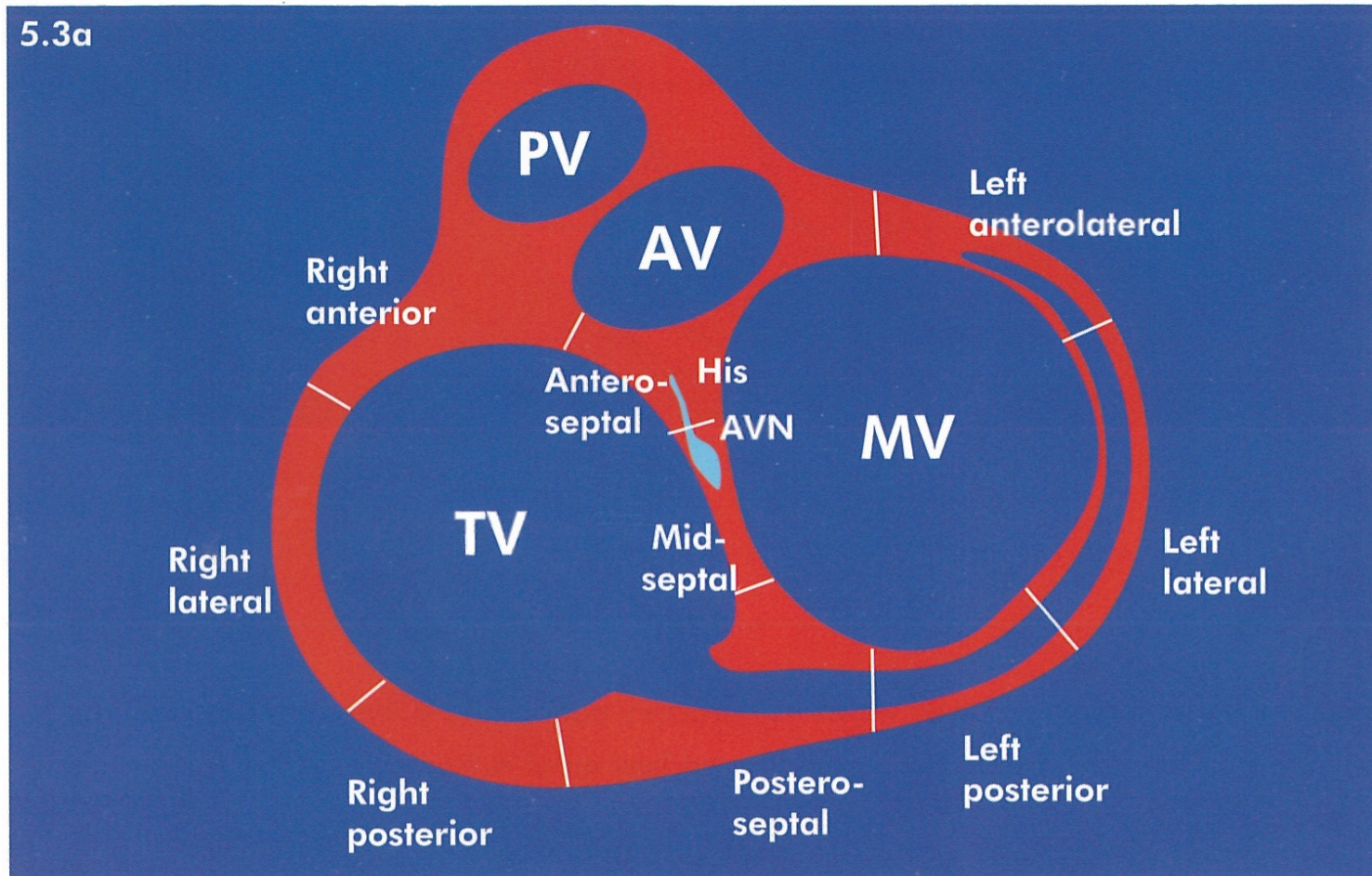
Base Pacing
With AP





Tracing 5.6 Pacing from the right ventricular apex (left panel) demonstrates earliest activation at the proximal CS with a stimulus-to-atrial interval of 140 ms. After the catheter is positioned at the RV base (right panel; Figure 5.6c), the stimulus-to-atrial interval shortens to 100 ms, which is consistent with the diagnosis of a right posterior accessory pathway.

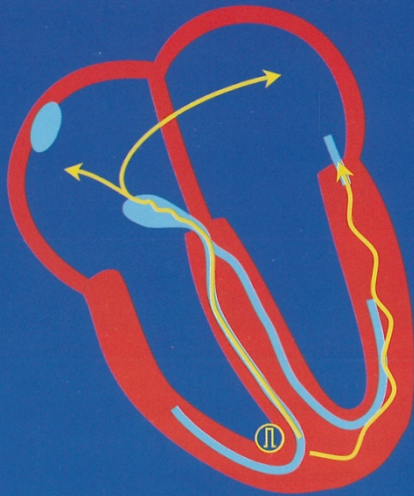
Location of Accessory Pathway



Retrograde Atrial Activation

Concentric vs Eccentric

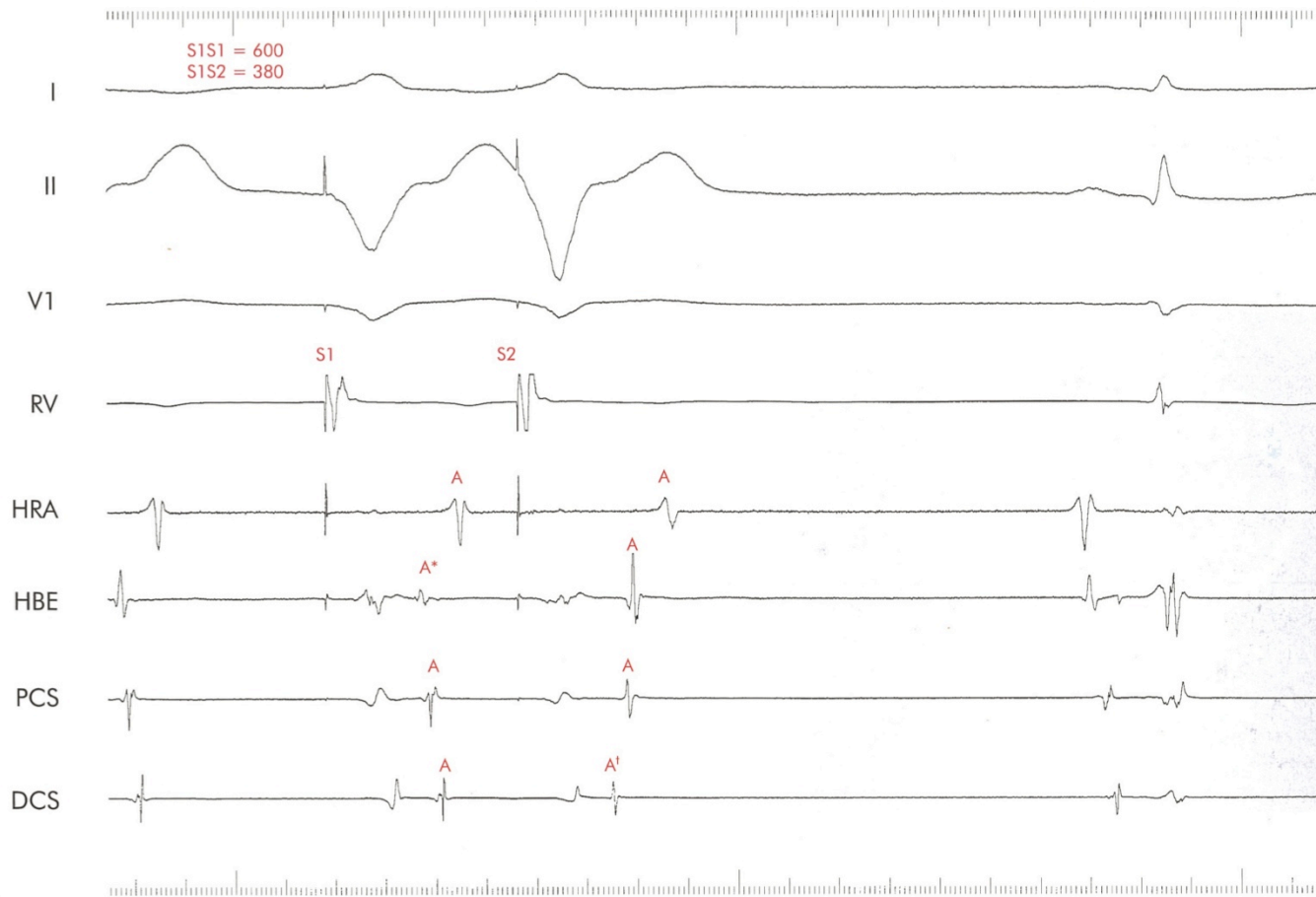
5.2a



5.2b

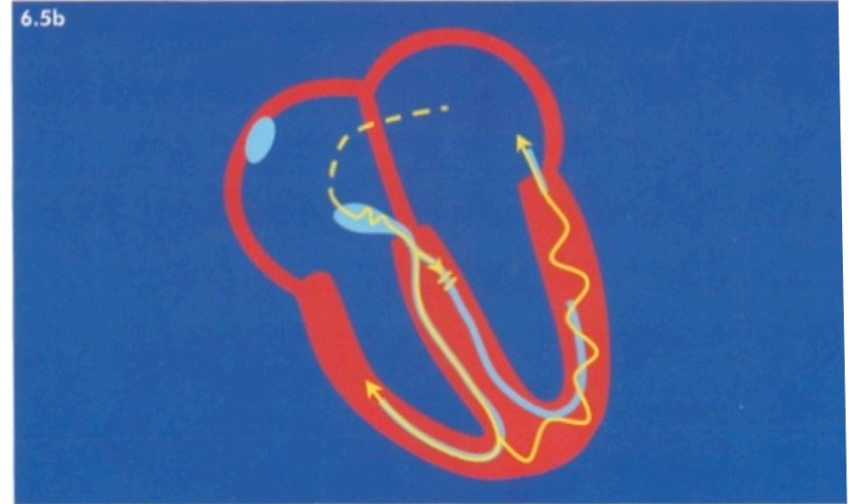
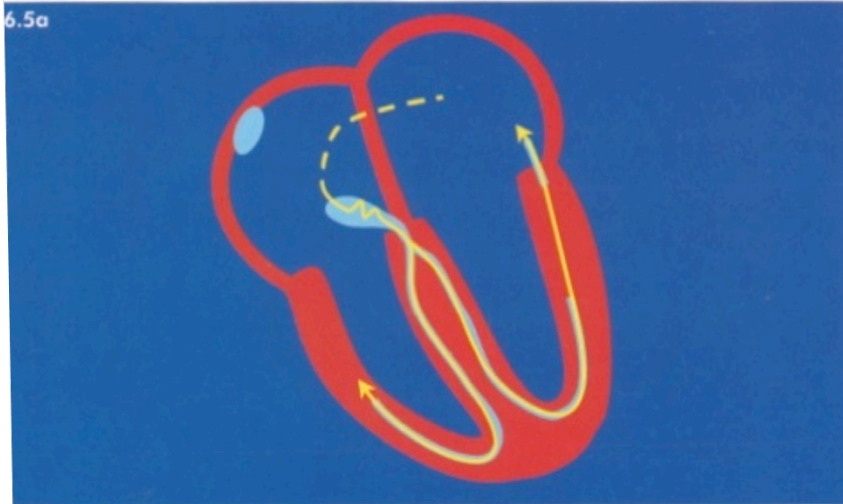


Retrograde Atrial Activation

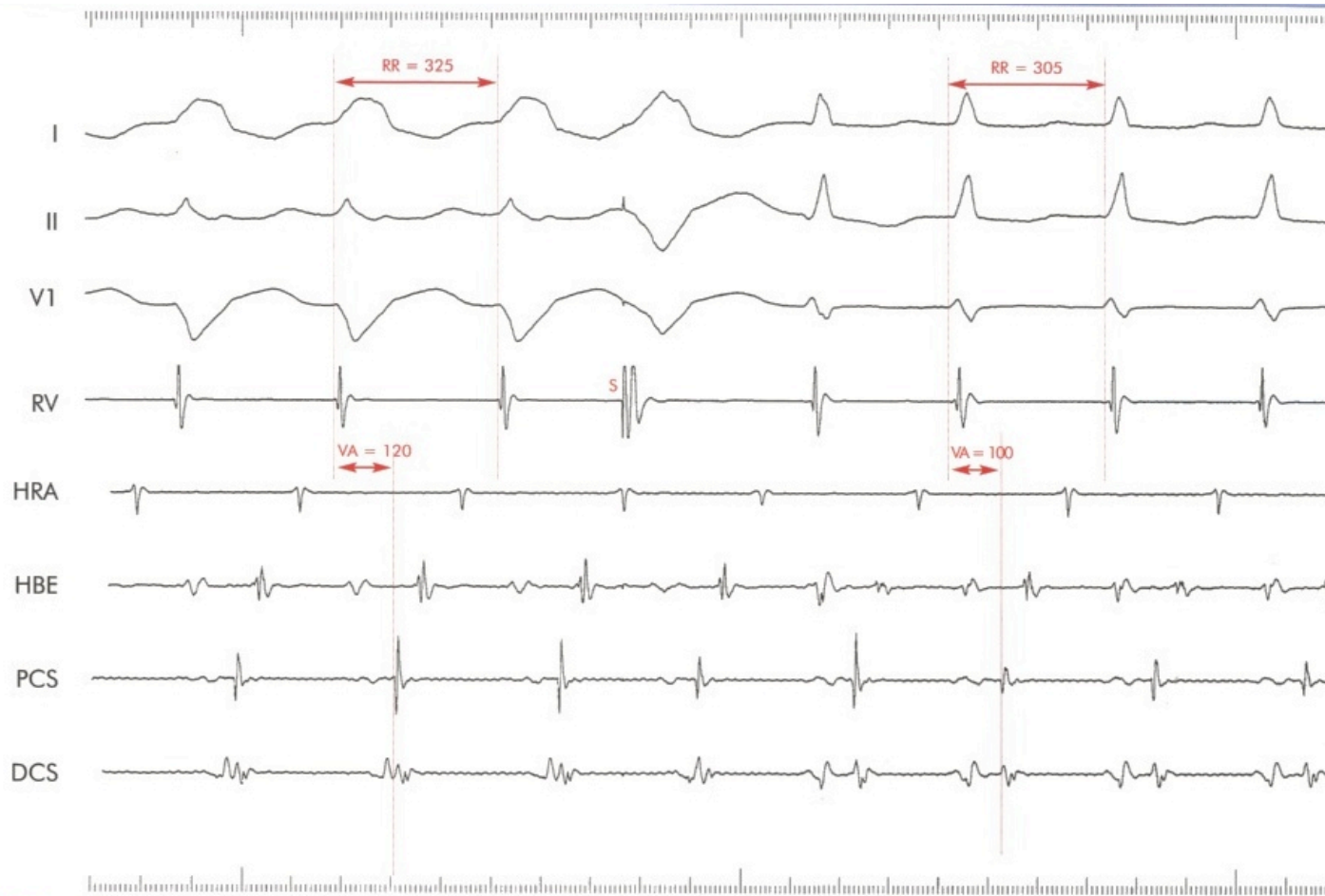


Tracing 5.2a A patient with a left free wall accessory pathway. During the ventricular drive train (S1), the atria are activated retrogradely by the His-Purkinje system and the AV node. The earliest atrial signal (A*) is recorded by the His catheter — indicating concentric atrial activation. A ventricular extrasystole (S2) with a coupling interval of 380 ms is delayed in the AV node, allowing the impulse to travel up the pathway to the atria. Atrial activation (A2) is now eccentric, with the earliest signal (A') recorded by the distal coronary sinus catheter.

Effect of BBB in SVT



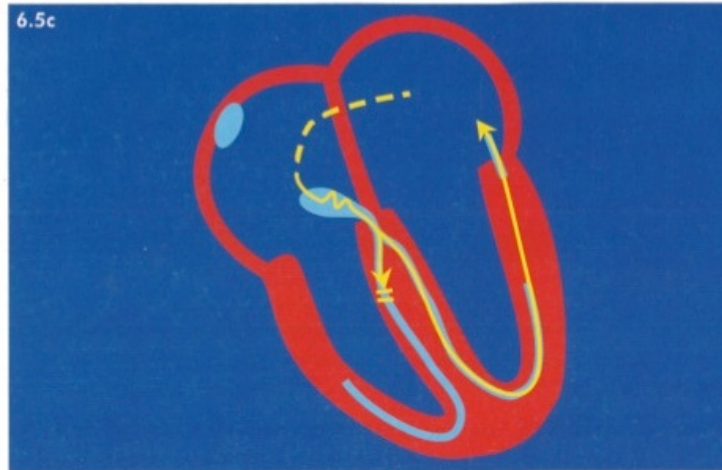
With LBBB the VA time increases



Tracing 6.5b AVRT with a left lateral accessory pathway. The first four beats show LBBB and the VA interval is 120 ms. Following a PVC (S), LBBB disappears and the VA interval shortens to 100 ms, with consequent shortening in the tachycardia cycle length. It is thus demonstrated that the left bundle branch participates in the circuit. Increase in tachycardia cycle length with bundle branch block is proof that the bundle, and by inference an ipsilateral accessory pathway, is part of the circuit.

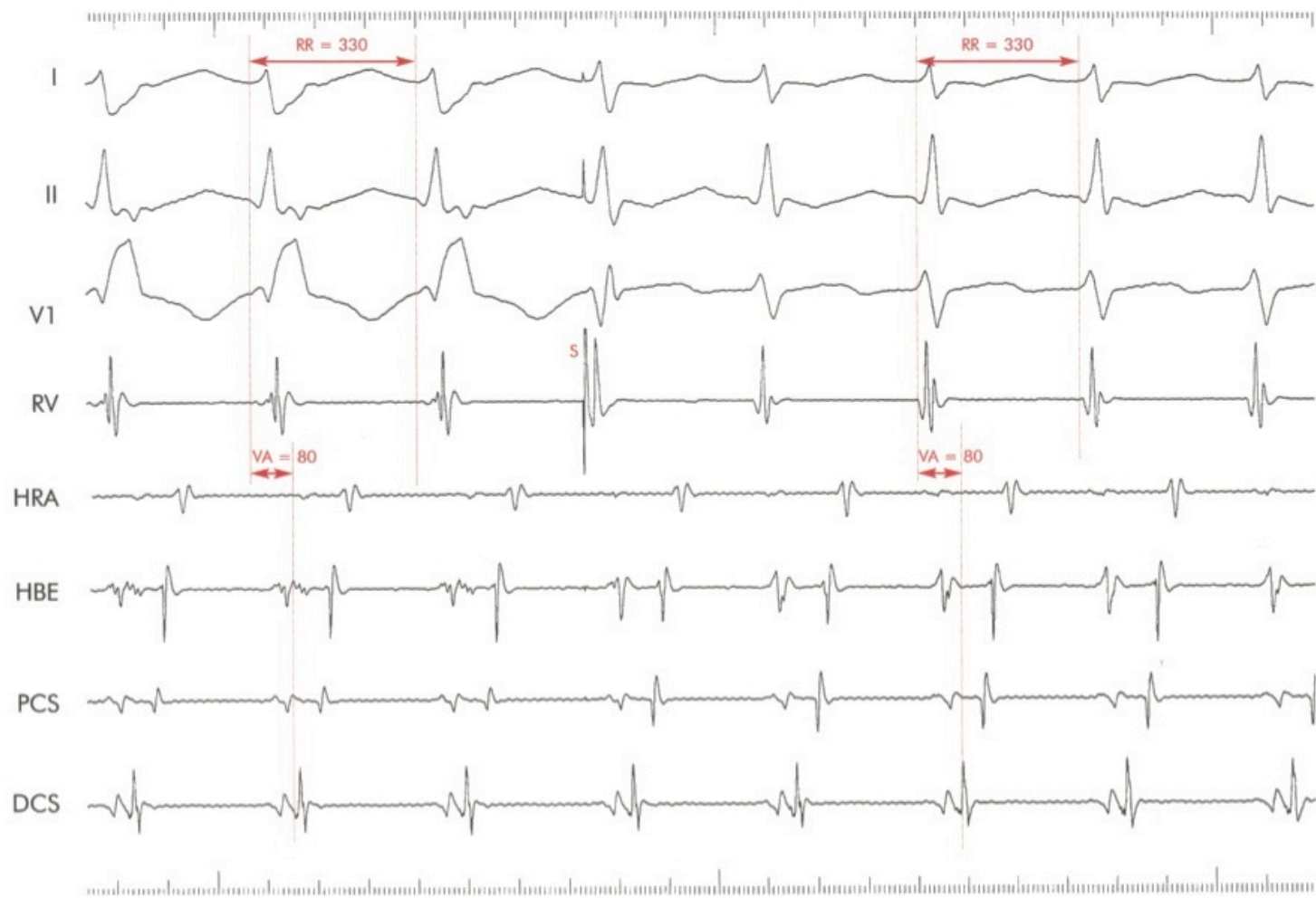
Note that the ventricular signals in the coronary sinus electrogram give a misleading impression of the VA interval. During LBBB, these signals arrive late compared to global ventricular activation, so, although the true VA interval is long, the local VA is short. The converse occurs when LBBB ceases. This illustrates the importance of measuring the VA interval from the very earliest ventricular activation (here, as is usual, the beginning of the surface QRS complex).

Left sided pathway and RBBB



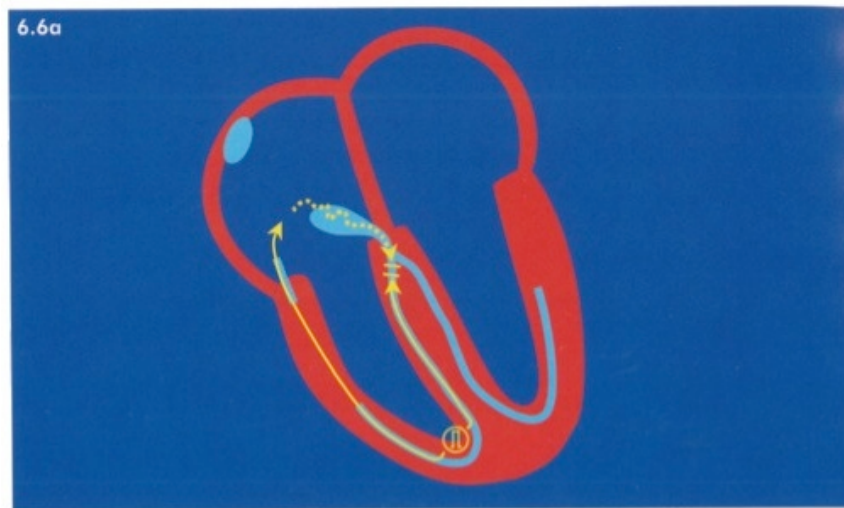
Left sided pathway and RBBB

No change in VA

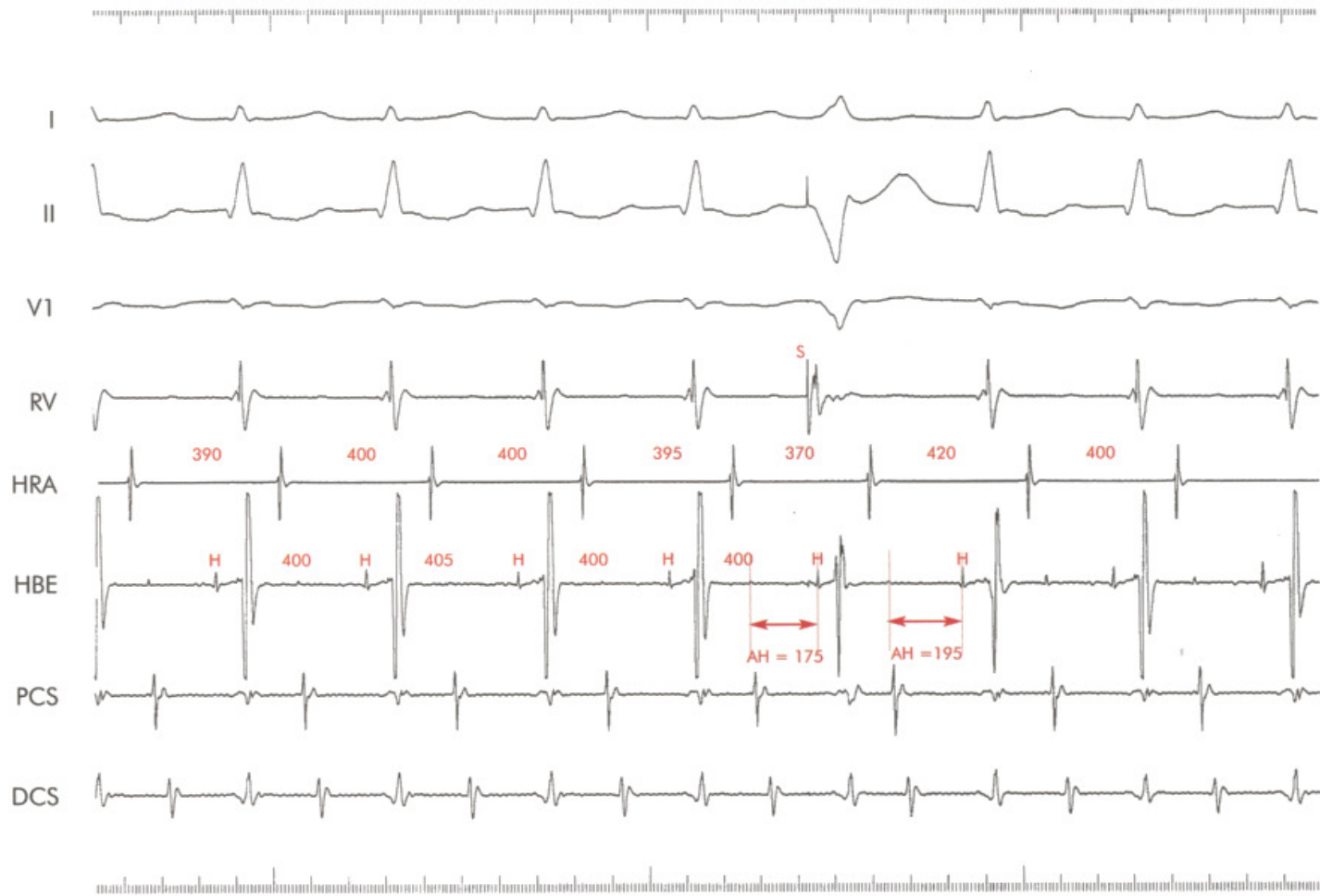


Tracing 6.5c AVRT with a left lateral accessory pathway. In the left half of the tracing, there is RBBB (most obvious in lead V1), which resolves with delivery of a single ventricular extrastimulus. This has no effect on the VA interval (and consequently none on the tachycardia cycle length), indicating that the right bundle branch (RBB) is not part of the circuit.

PVC on His



PVC on His

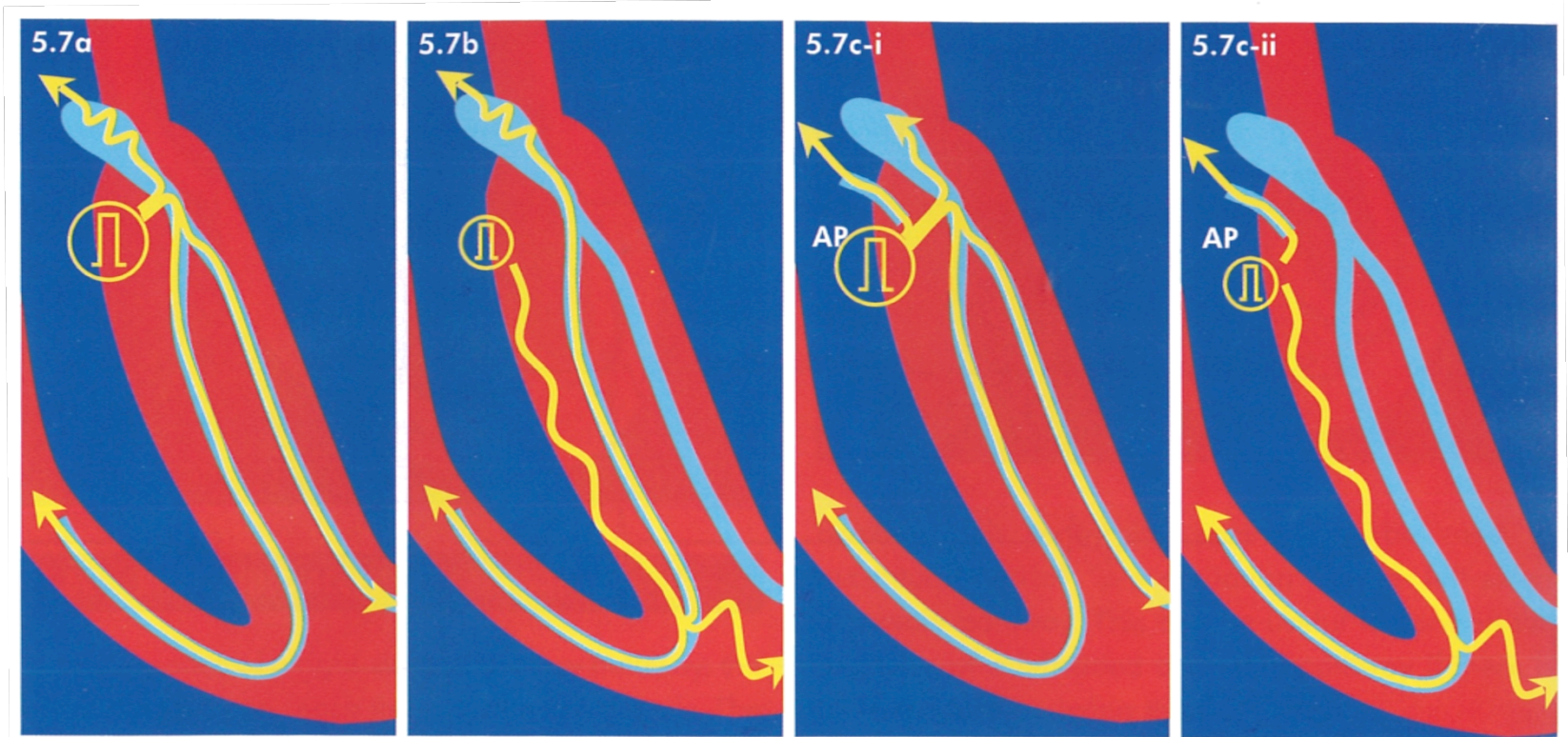


Tracing 6.6a AVRT with a right anterior accessory pathway. A single ventricular extrastimulus (S) is delivered just prior to the His deflection without affecting its timing. The result is advancement of the subsequent atrial electrogram by 30 ms, as measured in the high right atrium. An accessory pathway must therefore be present. Note that the tachycardia cycle length is very stable prior to the PVC; if there were significant 'wobble', it would be difficult to be certain that the advanced atrial electrogram was indeed due to the PVC. The early atrial activation results in an increased AH interval. As a result the whole circuit is only marginally advanced.

Parahisian Pacing

No AP

With AP



Tracing 5.7 On beats two and three there is His capture: the surface QRS complex is narrow and the RV apex is activated early. On beats one and four there is loss of His capture: the surface QRS complex is wide and the time from stimulus to RV apex activation is long. There is very little prolongation of the VA interval with loss of His capture. This indicates that retrograde conduction is not solely over the node, and that an accessory pathway close to the anteroseptal region is probably present. In this example, the earliest activation is in the proximal CS throughout, and the accessory pathway was ablated in the midseptal region. In the absence of an accessory pathway, loss of His capture would be accompanied by prolongation of the VA interval by at least 50 ms.

