ECG Pearls for Your Practice: Interactive Discussion

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Differential Diagnosis of ST-Segment Elevation

- Hyperkalemia
- Acute pericarditis
- Ventricular aneurysm
- Acute myocardial infarction
- Prinzmetal's angina
- Left ventricular hypertrophy
- Left bundle branch block
- Brugada syndrome
- Pulmonary embolism
- Cardioversion
- Normal (male-pattern)
- Early repolarization
- ST elevation of normal variant

Suggested article:	Abrahamian FM. ACS mimics: Non-AMI causes of ST-segment elevation. In: Matt A, Tabas JA, Barish RA, (eds). <i>Electrocardiography in Emergency Medicine</i> . ACEP; 2007:119
	131.

Wang K, Asinger RW, Marriott HJ. ST-segment elevation in conditions other than acute myocardial infarction. *N Engl J Med.* 2003;349:2128-2135.

Hyperkalemia

- Tall, narrow-based, and pointed T-waves
 - □ Earliest sign
 - □ Symmetrical and peaked T-waves (especially precordial leads)
 - □ "Tenting" or "peaking" with narrow base (amplitude of T-waves: > 6 mm in limb leads or > 10 mm in precordial leads)
- QT-interval shortening
- Prolongation of PR-interval
- Flattening (low amplitude) or absence of P-wave

- Widening of QRS complex
- May also see ST-segment elevation (often downsloping) or depression
- Sine-wave
- Altered cardiac conduction (can cause any type of a block)
- Relationship between serum K⁺ and ECG changes vary among different patients
- Not a reliable test for mild (5.5-6.5) hyperkalemia
- ECG changes typically start around K⁺ of 6.8

Suggested article:	Mattu A, Brady WJ, Robinson DA. Electrocardiographic
	manifestations of hyperkalemia. Am J Emerg Med. 2000;18:721-
	729.

DDx of Conditions that Can Cause Peaked T-wave

- Hyperkalemia
- Early acute MI
 - □ T-waves are broad rather than narrow and pointed and often associated with long QT-interval

Note: Intracranial hemorrhage can be associated with deep inverted T waves

- **D** Other associated findings are prolonged QT-interval, prominent U-wave
- Commonly seen in precordial leads

<u>T-wave</u>

- Normal T-wave has an initial slow phase followed by a fast phase
- When you divide the T-wave in half, the area under the curve is **not** symmetrical
- T-wave usually is $\geq 10\%$ the height of the R-wave
- Always inverted in aVR
- Always upright in leads I, II, and V4-V6
- Usually same direction as QRS complex except in right precordial leads (V1,V2)

<u>U-wave</u>

- Normal U-wave has an initial fast phase followed by a slow phase (opposite to T-wave)
- Upright in all leads except in aVR
- Follows T-wave axis
- Usually < 1.5 mm and is 5-25% height of the T-wave
- Largest and best seen in leads V2 and V3
- Prominent U-wave: Amplitude > 1.5 mm
- DDx of prominent U-wave: Hypokalemia, hypothermia, bradyarrhythmias, intracranial hemorrhage

Pericarditis

• Stages:

		Stage 1:	PR-segment depression Best seen in lead I Precedes ST-segment elevation
			Widespread ST-segment elevation (seldom exceeds 5mm) Concave upward No reciprocal depression
			Reverse findings in lead aVR: PR-segment elevation and ST- segment depression
		Stage 2:	PR-segment and ST-segment returns to baseline T-wave amplitude begins to decrease
		Stage 3:	Inverted T-waves
		Stage 4:	Normal ECG
,	Us	e TP-segment a	as your baseline

- Look at lead aVL:
 - □ The ST-segment elevation in patients with infarction behaves reciprocally between leads III and aVL
 - □ The ST-segment in patients with acute pericarditis does not result in ST-depression in aVL
- Look at V6 to differentiate acute pericarditis from early repolarization:
 - □ Acute pericarditis: Ratio of ST-segment (mm) to T-wave amplitude (mm) ≥ 0.25
 - □ Early repolarization: Ratio of ST-segment (mm) to T-wave amplitude (mm) < 0.25
- DDx of PR-segment depression: Acute pericarditis, atrial infarction, and early repolarization, pericardial effusion/cardiac tamponade

Suggested article: Lange RA, Hillis LD. Acute pericarditis. *N Engl J Med.* 2004;351:2195-2202.

Ventricular Aneurysm (Dyskinetic Ventricular Segment)

- More common in men (men: female ration of 4:1)
- Commonly seen with transmural myocardial infarction
- 80% are located anterolaterally and are associated with total occlusion of left anterior descending artery
- Inferior/posterior aneurysms are less common
- Other causes of left ventricular aneurysm are blunt chest trauma, Chagas disease, sarcoidosis
- Amount of ST-segment does not correlate with the size of left ventricular aneurysm
- QRS duration increase with the age of the aneurysm
- Characteristic features on ECG:
 - □ Old infarction (large Q-waves) with persistent ST-segment elevation
 - □ ST-segment elevation with varying morphologies; commonly concave. If non-concave, suspect myocardial infarction.
 - \Box ST-segment elevation is often < 3 mm and usually does not extent into lead V5
 - □ No reciprocal changes
 - **Q** waves in the same distribution of ST-segment elevation
 - Q waves can appear as early as 2 hours after myocardial infarction
 - Remember the rule of 80:20: In 80% of cases Q waves appear within 8 hours and in 20% of cases Q waves appear within 2 hours
 - □ Loss or poor R-wave progression
 - □ No change with serial ECGs or intervention (no dynamic changes)
- Diagnose: Echocardiography (sensitivity 93%; specificity 94%) Cardiac catheterization (gold standard)

Suggested article:	Engel J, Brady WJ, Mattu A, et al. Electrocardiographic ST-
	segment elevation: Left ventricular aneurysm. Am J Emerg Med.
	2002;20:238-242.

Acute Myocardial Infarction (AMI)

- ST-segment with a plateau or convex shape
- A concave shaped ST-segment elevation does not rule out AMI
- Look for reciprocal behavior (especially between leads aVL and III)
- Reciprocal changes can be absent in $\sim 20\%$ of the time
- Q-waves can develop as early as 2-4 hours
- Most develop within 8 hours
- With inferior wall MI, look for right ventricular infarction
 - □ Clues: Look for ST-segment elevation in V4R and V1
 - \Box ST-segment elevation of > 1 mm in lead V4R with an upright T-wave in the same lead is the most sensitive electrocardiographic sign of right ventricular infarction

ECG Manifestations of AMI with Corresponding Reciprocal Changes

Location	ST segment elevation	Reciprocal changes (ST-segment depression)
Inferior	II, III, aVF	I, aVL or V1-V2
Anteroseptal	V1-V4	II, III, aVF
Lateral	V5,V6, I, aVL	V1,V2
Right ventricle	V4R	
Posterior	V8,V9	V1,V2

Prinzmetal's Angina

- The ECG manifestations of Prinzmetal's angina and AMI are indistinguishable
 - With Prinzmetal's angina, the ST-segment elevation is transient
 - □ Prolonged spasm can cause infarction

Left Ventricular Hypertrophy (LVH)

- One of the conditions frequently mistaken for acute infarction
- ST-segment:
 - □ Seen in precordial leads V1-V3 (often < 2 mm)
 - □ Concave shaped
 - **D** The deeper the S-wave, the greater the ST-segment elevation
- Various voltage and non-voltage related ECG criteria exist for LVH with variable sensitivities (voltage criteria only 30% sensitive)
- Scoring system (e.g., Romhilt and Estes criteria) combining voltage and non-voltage related ECG findings associated with LVH increase sensitivity

Voltage Criteria for LVH:

- Cornell criteria (most accurate):
 R-wave in aVL + S-wave in V3
 - \Rightarrow > 28 mm in males
 - 20 mm in females

Examples of Other Voltage Criteria for LVH:

- Precordial leads:
 - **\Box** R-wave in V5 or V6 + S-wave in V1
 - ♦ \geq 35 mm if age \geq 20 years
 - ♦ \geq 45 mm if age < 20 years or with left bundle branch block
- Limb leads:
 - □ R-wave in aVL ≥ 12 mm (a highly specific finding)

Non-voltage Related Findings Associated with LVH:

- ST-segment and T-wave changes (secondary ST-T changes) also known as "strain pattern"
 - ST-segment and T-wave deviation opposite in direction to the major deflection of QRS
 - □ ST-depression with T-wave inversion in leads I, aVL, V5, V6
 - □ ST-segment often downsloping (hockey stick shape)
 - Consider ischemic process if associated with horizontal ST-segment depression
 - □ T-waves are asymmetrical (slow downward phase with fast upward wave) and **not** deep
 - Consider ischemic process if associated with deep symmetrical inverted Twaves
 - Classic ST-T changes are usually found in patients with fully developed LVH
- Left atrial enlargement
- Left axis deviation
- Widened QRS complex
- Delayed intrinsicoid deflection (> 0.04 seconds) in left chest leads (but remains normal in right chest leads)

Left Bundle Branch Block (LBBB)

- The abnormal ventricular depolarization as well as secondary ST-T changes makes the diagnosis of concomitant AMI in the presence of LBBB difficult
- Normally, in LBBB the ST-segment and the T wave act in a discordant fashion with the main QRS complex
 - □ If the main QRS complex is positive (e.g., in leads I, aVL, V5, and V6), then the expected secondary ST-T changes will be ST-segment depression with T wave inversion
 - □ If the main QRS complex is negative (e.g., in leads V1 and V2), then the expected secondary ST-T changes will be concave ST segment elevation with upright T wave
- In LBBB, the presence of concordant changes (i.e., ST-segment elevation ≥1 mm in leads with a positive QRS complex such as lead V5, or ST-segment depression ≥1 mm in leads with a negative QRS complex such as leads V1-V3, II, III, aVF) are abnormal and considered highly specific and predictive for myocardial infarction.

- □ However, the limitation of these ECG findings lies with its low sensitivity and poor negative likelihood ratio; hence, absence of these features cannot be used to exclude patients with AMI.
- Another ECG feature suggestive of AMI in the presence of LBBB is extreme (i.e., ≥5 mm) discordant ST-segment deviation. Similarly, this ECG feature also exhibits low sensitivity and may be present in the absence of acute infarction.
- Additional ECG features suggestive of myocardial infarction with LBBB may include: replacement of the secondary concave ST-segment elevations with a convex ST-segment; deep T wave inversion in leads V1 to V3; the presence of Q waves in at least two of the leads I, aVL, V5, or V6; and Q waves in II, III, and aVF especially if associated with T wave inversions.
- Clues to prior myocardial infarction may also include notching of the upstroke part of a wide S wave in at least two of the leads V3, V4, or V5 (the Cabrera sign), or notching of the R wave upstroke in leads I, aVL, V5, and V6 (the Chapman sign).
- Obtaining serial ECGs looking for dynamic changes, as well as comparison to previous ECGs are also invaluable in identifying patients with acute pathology

Suggested articles:	Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. <i>N Engl</i> <i>J Med.</i> 1996;334:481-487. [Erratum in: <i>N Engl J Med.</i> 1996;334:931].
	Li SF, Walden PL, Marcilla O, et al. Electrocardiographic diagnosis of myocardial infarction in patients with left bundle

branch block. Ann Emerg Med. 2000;36:561-565.

Brugada Syndrome

- Accounts for 40%-60% of all cases of idiopathic ventricular fibrillation
- The syndrome has been linked to mutations in the cardiac sodium-channel gene
- Depression or a loss of the action-potential dome in the right ventricular epicardium
- The ST-segment elevation associated with Brugada syndrome is limited to leads V1-V2 or V3.

- Typically, it has a saddleback or coved appearance with a gradual downslope, ending with an inverted T wave
- The high take-off ST-segment in V1-V2 resembles the rSR' pattern seen with RBBB. However the wide S wave in leads I, aVL, and V6 that are associated with RBBB may be absent in Brugada syndrome. Most often the QT interval is within normal limits and the PR interval is prolonged.
- The terminal portion of the QRS complex and the beginning of the ST-segment is indistinct. In contrast, the ST-segment associated with anteroseptal infarction complicated by RBBB has a distinct transition from the QRS complex with a horizontal or upsloping (convex), rather than downsloping, morphology.
- The ultimate diagnosis rests on exclusion of other conditions resulting in ST-segment elevation in the right precordial leads (e.g., early repolarization, LBBB, LVH, or AMI), electrophysiological studies, or with the aid of a pharmacological challenge. Arrhythmogenic right ventricular cardiomyopathy also has a similar ECG pattern to that of Brugada syndrome and the ECG distinction is difficult. A drug challenge with sodium channel blockers may help in differentiating these two conditions.

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Suggested article: Antzelevitch C, Brugada P, Borggrefe M, et al. Brugada syndrome:
Report of the second consensus conference. Circulation. 2005;111:
659-670.
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Pulmonary Embolism (PE)

- Incidence and severity of the ECG pattern depends on the timing and magnitude of the obstruction in the pulmonary vasculature
- > 20 different ECG manifestations of PE have been discussed in medical literature
- ECG not useful and/or sensitive for diagnosis
- Most are nonspecific findings and often transient
- Sinus tachycardia is the most frequent rhythm disturbance
- Most frequent ECG pattern: Sinus tachycardia with non-specific ST segment/T wave changes
- Other findings:
 - □ Atrial arrhythmias (a.fib/flutter)
 - **□** Right bundle branch block (complete or incomplete)
 - □ Right-axis deviation or left-axis deviation (LAD occurs more often due to preexisting disease)
 - □ Tall, peaked P-wave with amplitude > 2.5 mV in lead II (P-pulmonale)

- \Box S₁Q₃T₃ (not pathognomonic; not sensitive; seen in < 30%)
- Right ventricular strain pattern (ST-segment depression with inverted T-wave in V1 and V2
- May be associated with ST-segment elevation in the inferior and to lesser degree in anteroseptal leads ("pseudoinfarct pattern")
- Inverted T-waves V1-V3 (common finding in massive PE and is the most persistent of all ECG abnormalities)

Cardioversion

- Transient ST-segment deviations, either depressions or elevations, can be encountered with transthoracic and epicardial electrical shocks
- The ST-segment elevation at times could be significant (> 5 mm), but it only lasts one to three minutes after the cardioversion
- In comparison to the patients without ST-segment elevation, patients with STsegment elevation often have a lower conversion rate and are less likely to remain in sinus rhythm
- The mechanism of ST-segment elevation associated with cardioversion is not well understood

Normal ST-Segment Elevation

- A majority of healthy men will commonly display ST-segment elevation in the precordial leads V1-V4
- The prevalence of this so-called "male pattern" ST-segment elevation is very common and is considered to be a normal finding. It is highest in the age group of 17 to 24 years and declines gradually with advancing age.
- The amplitude of the ST-segment elevation ranges from 1-3 mm (most marked in V2) with a concave morphology. There are no associated T wave abnormalities or reciprocal changes.
- Similar ST-segment elevation is less frequently observed in women. If present, the ST-segment elevation in "female pattern" is most commonly < 1 mm.

Early Repolarization (Normal Variant)

- A commonly observed normal variant, often referred to as early repolarization, is also associated with ST-segment elevation in the precordial leads (most commonly involving leads V2-V5)
- The amplitude of the ST-segment elevation ranges from 1-4 mm (most marked in V4) with a concave morphology
 - Other associated findings include a notch at the J point and tall, upright T waves. There are no reciprocal changes.
- Less commonly, early repolarization can involve the limb leads. In this case, the associated findings include ST-segment elevation in limb leads (commonly observed in the inferior leads II, III, aVF with ST-segment elevation in lead II > lead III) and reciprocal ST-segment depression in aVR.
- Early repolarization can also involve the atrial tissue which manifests as PR segment depression. The ECG changes associated with early repolarization at times can be confused with ECG changes of stage 1 pericarditis.

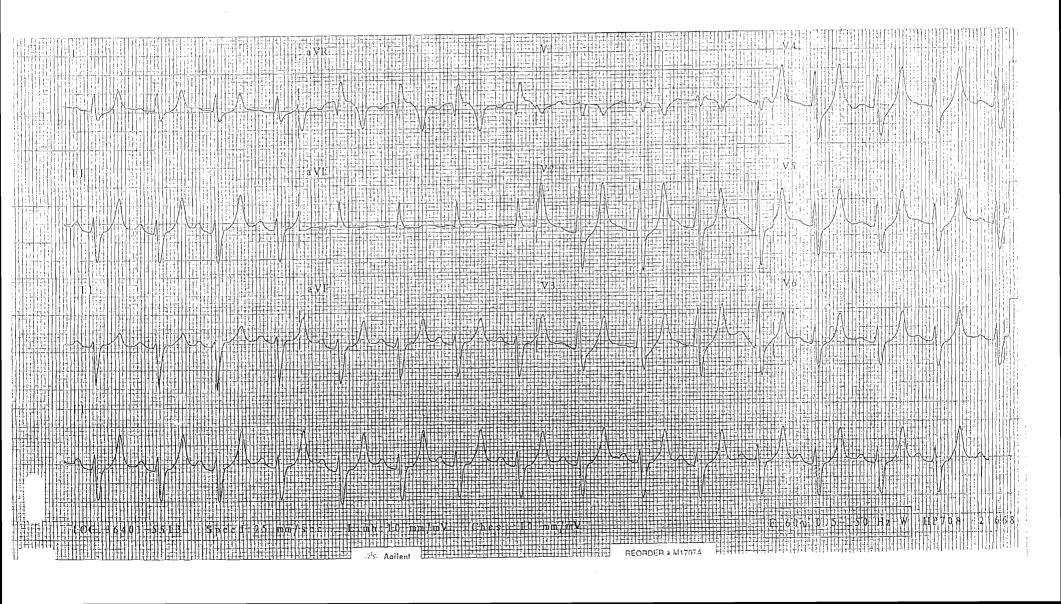
ST Elevation of Normal Variant

- Midprecordial (leads V3-V5) ST-segment elevation with terminal T wave inversion can also be a normal finding. This is referred to as "ST elevation of the normal variant" and often seen in young black men.
- The morphology of the ST-segment tends to be concave. Other associated findings include short QT interval and high QRS voltage.
- Differentiating this variant from AMI may be difficult
- Helpful clues favoring myocardial ischemia may include convex ST segment elevations, a prolonged QT interval and deep, symmetrical T wave inversions

Take Home Points

- Hyperkalemic T wave: Tall with narrow base
- Hyperacute T waves: Tall with broad base
- Pericarditis: Look at lead V6 for ST-segment / T-wave amplitude ≥ 0.25
- ST elevation with infarction behaves reciprocally between leads III and aVL
- Q waves + ST elevations in V1-V4: Think left ventricular aneurysm (LVA)
- LBBB: ST-segment and T wave act in discordant fashion with the main QRS complex
- Always get serial ECGs

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Т	61	Unauthorized use is promoticu. Preliminary-md must review	
QRS	-83	Unauthorized use is prohibited. preliminary-md must review	
Р	79		
AX I	[S – –	3	
•			
QTc	465		
QT	374		
QRSD	124	. Bifascicular block: RBBB & LAFB	
PR	196	. Borderline first degree AV block	
Raie	93	. Sinus rhythm, rate 93	



. Normal sinus rhythm, rate 94......Normal P axis, PR, rate & rhythm Rate 94 \$ Leftward axis, probably normal for age.....QRS axis -15 to -30 & age > 40 PR 161 QRSD 91 consider posterior infarct 347 OT QTc 434 --AX1S-р 63 Unauthorized use is prohibited. QRS -29 PRELIMINARY-MD MUST REVIEW 53 Т VS

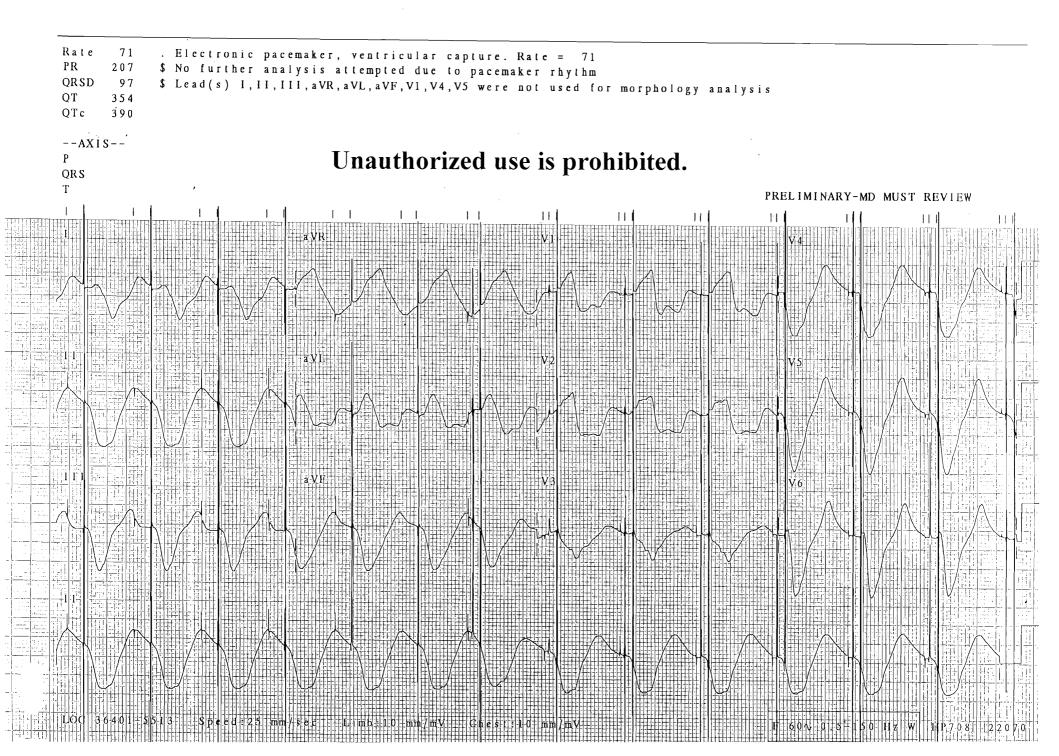
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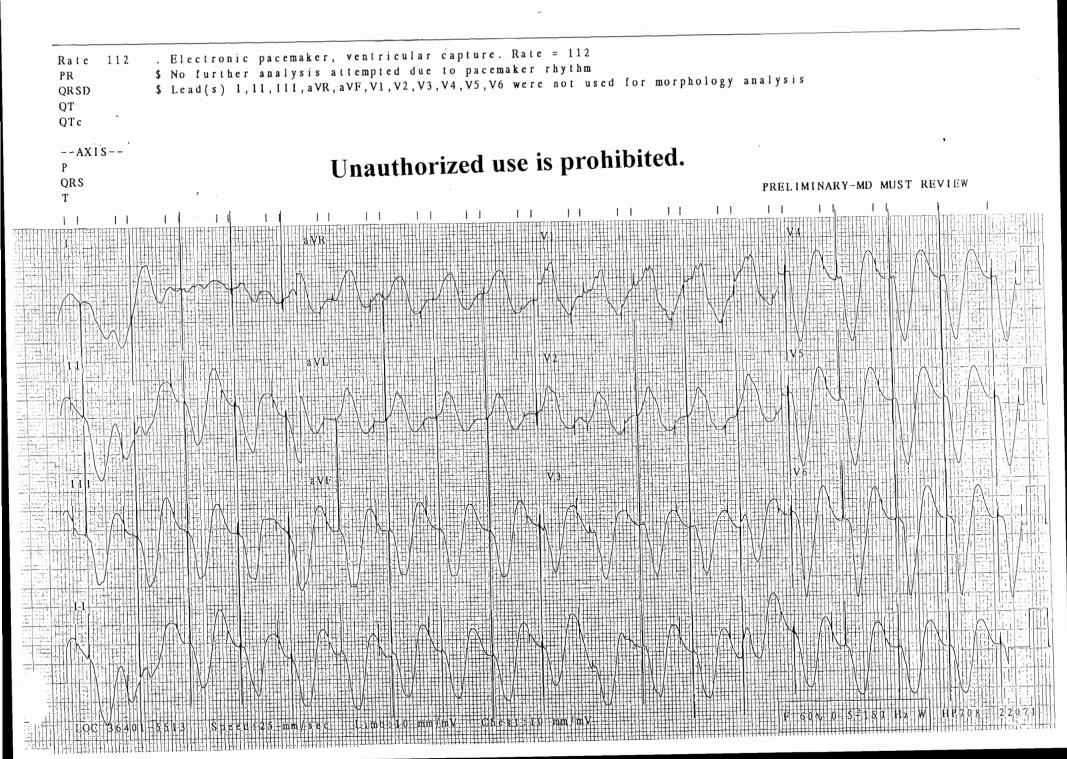
LOC 36401-5538 Speed:25-mm/sec = Linc:10-mm/mV Chest:10 mm/mV

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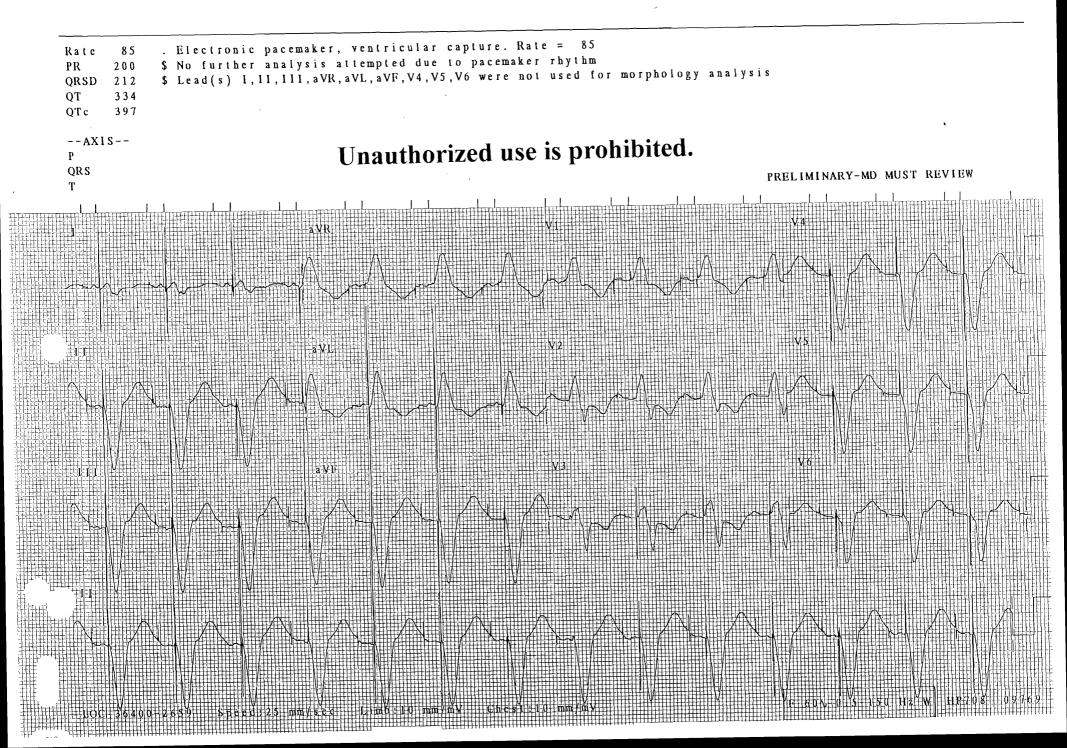
63 years. Male

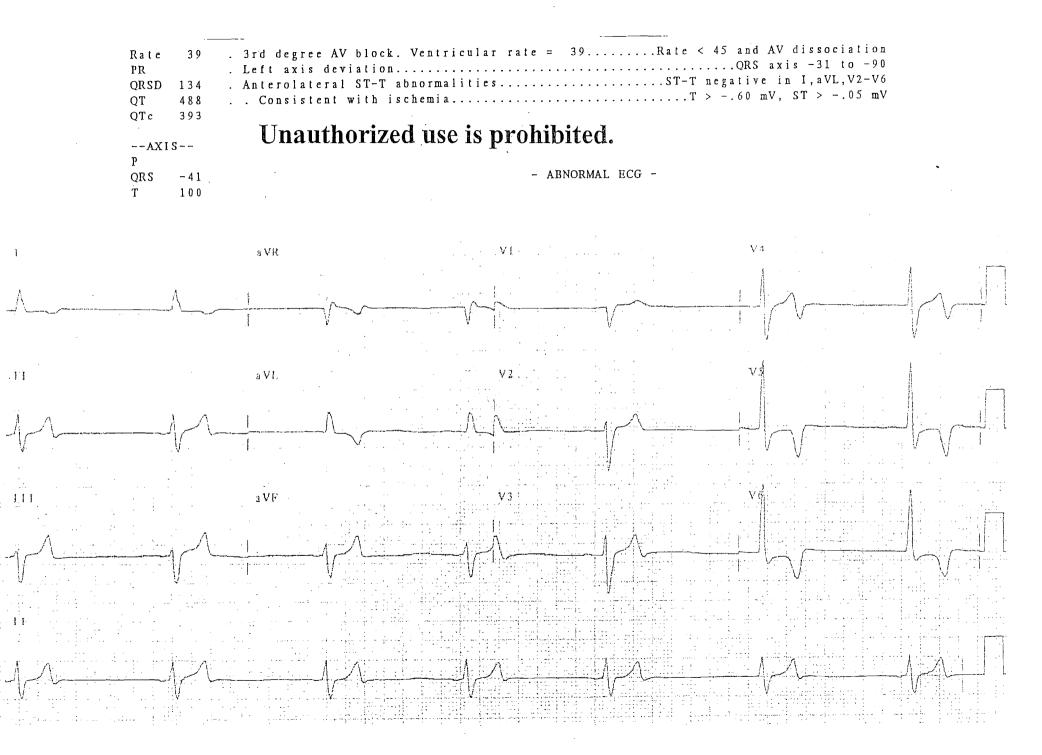
Rate 68 . Normal sinus 'rhythm, rate 68.....Normal P axis, PR, rate & rhythm PR 169 . Low voltage throughout..... Inv. Limb < 0.5 mV, chest < 1.0 mV QRSD 64 . Inferior infarct, age indeterminate......Q's & neg T's II, III, aVF OT 364 OTC 387 --AXIS---17 Р ORS -28 - ABNORMAL ECG -- 4 2 PRELIMINARY-MD MUST REVIEW VENNALL MENITONCE



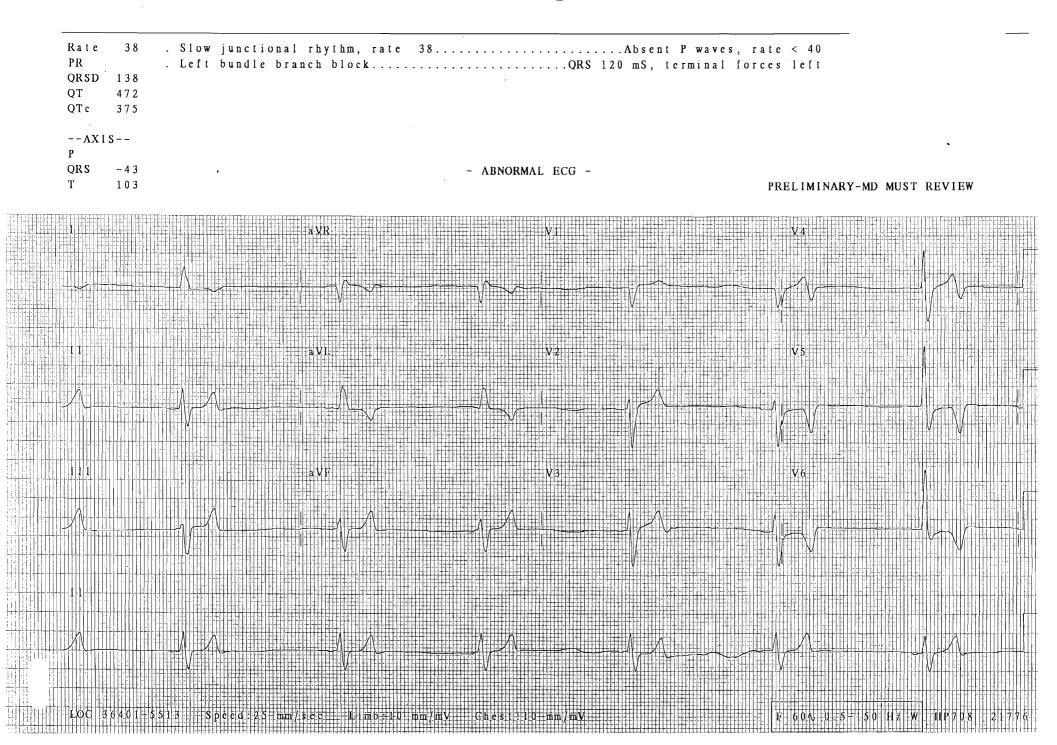


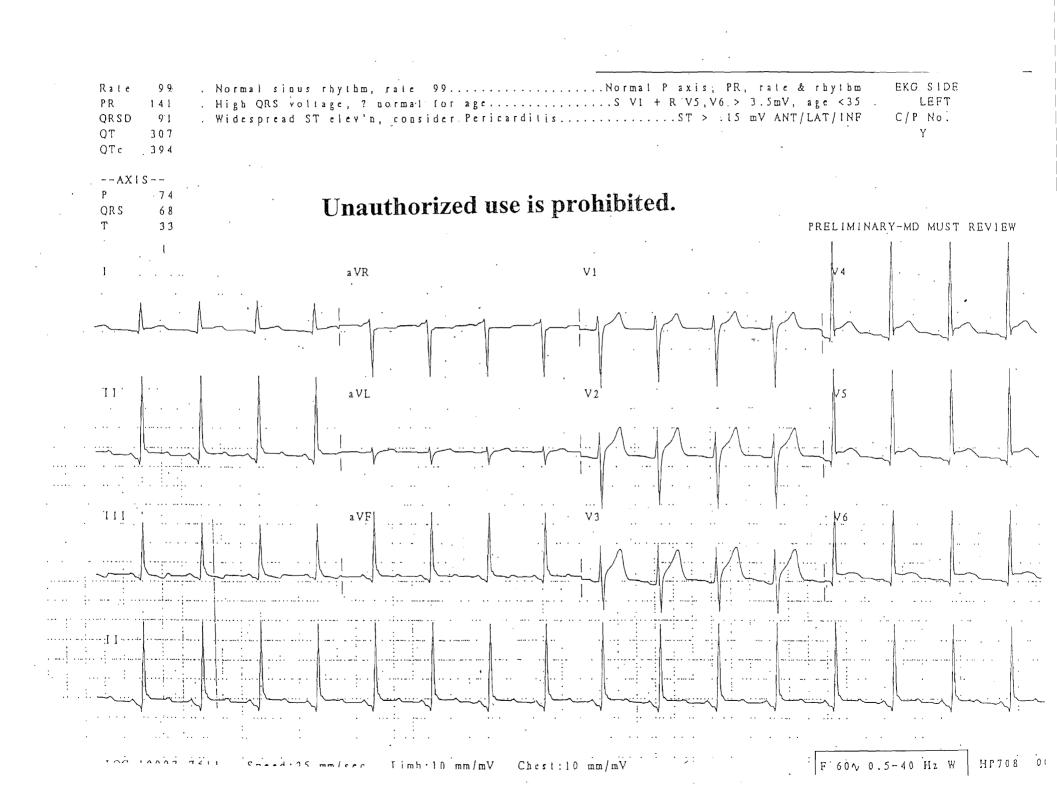
06/23/2003 10:58:51 AM 74 years Female





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Rate PR QRSD QT	73 149 95 314	. Normal s . QT inter . Widespre	inus 1 val sl ad ST	rhythm, rate 73 hort for rate elevation,consider	Pericarditi	7Normal P 	axis, PR, ra C .ST > .15 mV	ate & rhythm QTc < 360 mS ANT/LAT/INF		·	
QTc AX	346								Reque	sted by:	
P QRS	38 67	-		TT ()			J				
Т	53			Unauthorize	a use is p	oromotie	d o	PRELIMI	NARY-MD M	UST REVIE	εw

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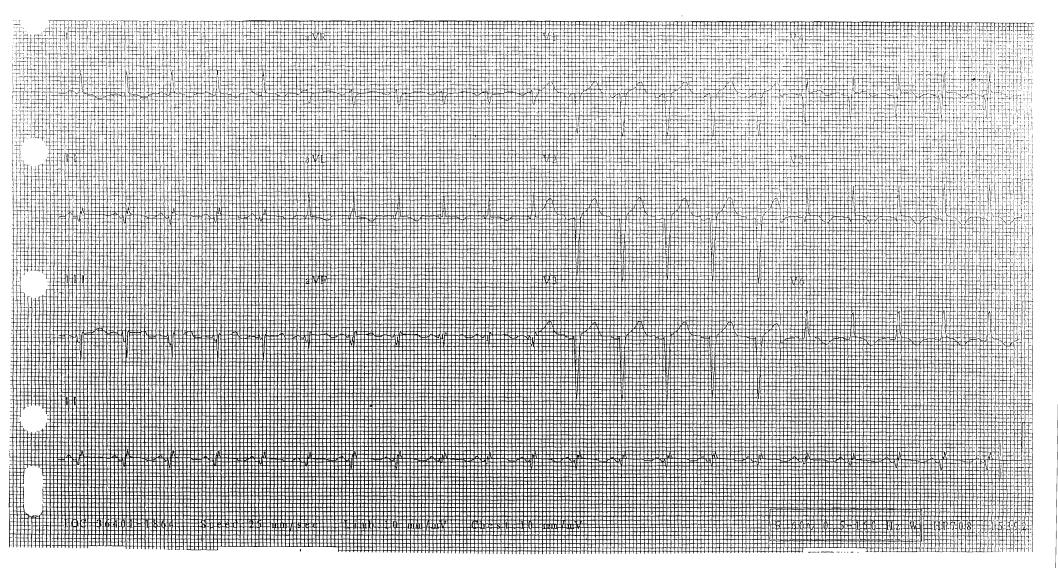
Rate 125 . Consider left atrial enlargement...... P V1 -.10 mV or more negative PR 115 . Anterior infarct, age indeterminate.....Q waves V2-V4, neg T's QRSD 85 . Nonspecific Lateral T wave abnormalities......T waves -.20 mV 1, aVL, V5, V6 QT 285 QTc 411 . . Cannot exclude ischemia......T > -.20 mV Requested by: . Borderline ST elevation, inferior leads.....ST > .07 mV II, III, aVF

--AXIS--

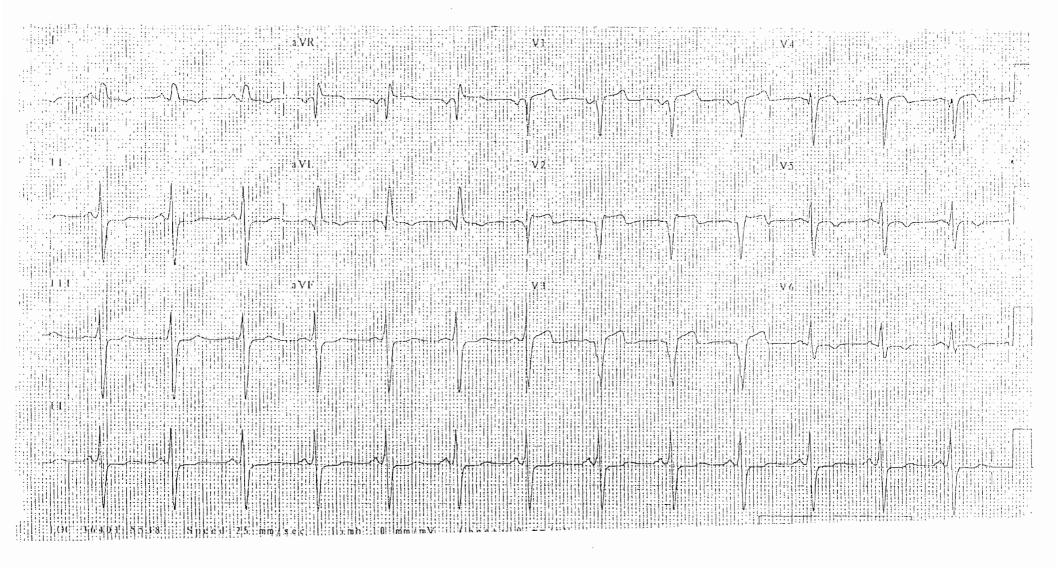
P 59 QRS -4 T 128

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PRELIMINARY-MD MUST REVIEW



Rate	.81	. Normal sinus rhythm, rate 81
PR	134.	. Left anterior fascicular block andQRS axis -45 deg., QRS > 120 mS
QRSD	124	nonspecific intraventricular conduction
QT	401	
QTc	465	. Left atrial enlargement Requested by:
		. Anterior infarct, possibly acuteQ waves V2-V4, ST +.15 mV
AX I	S – –	. Nonspecific Lateral T wave abnormalities
Р	36	
QRS .	-60	
T	129	Unauthorized use is prohibited. PRELIMINARY-MD MUST REVIEW

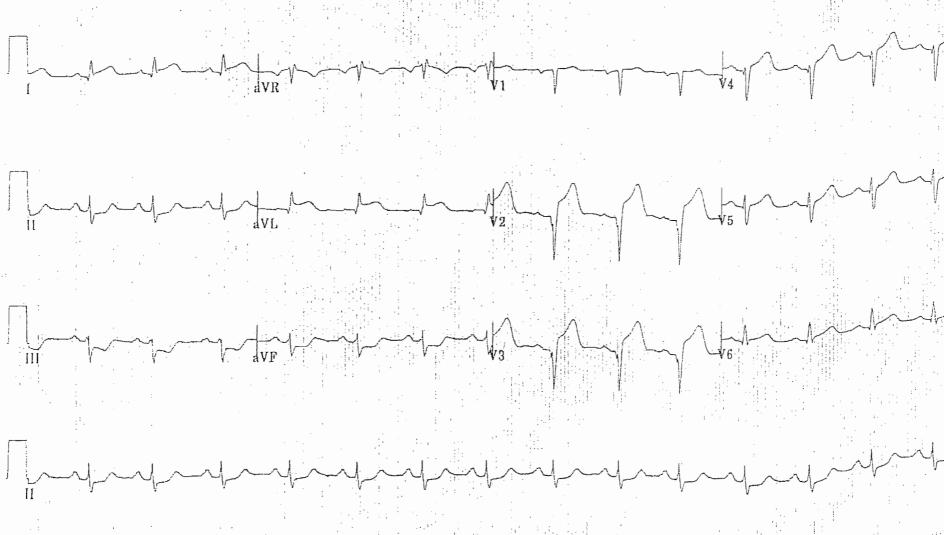


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	~ •

62years		Vent. rate	85 bpm
Male	Caucasian	PR interval	170 ms
		QRS duration	92 ms
Room: M2		QT/QTc 352	2/418 ms
		P-R-Taxes 5	7 -26 -6

Technisian: Test inl: Normal sinus rhythm Anteroseptal infarct, possibly acute Lateral injury pattern ** ** ** ** Acute MI * ** ** ** Abnormal ECG

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150 Hz 25.0 mn/s 10.0 mm/mV

4 by 2.5s + 1 rhythm ld

MAC 8 001G 12SLtm v250



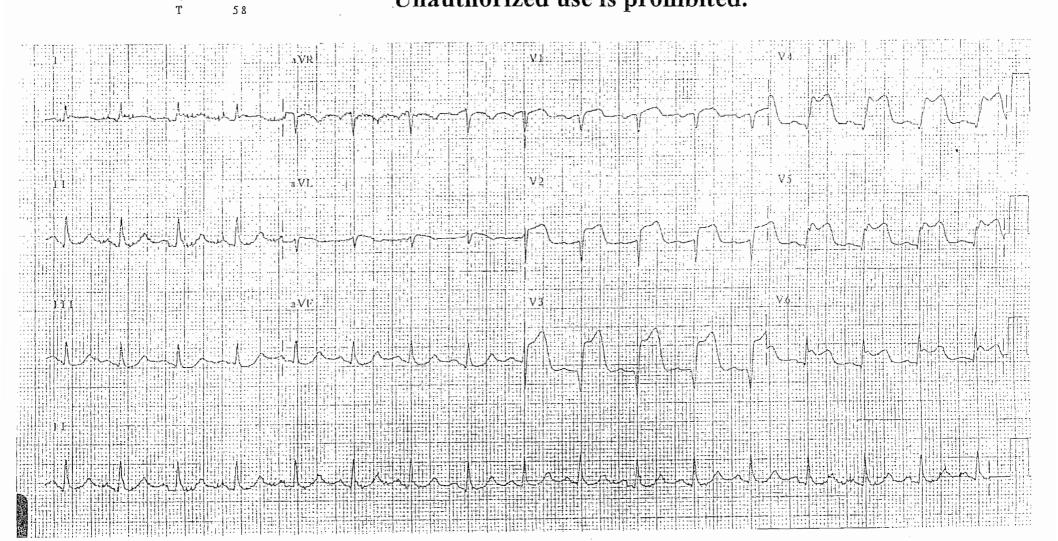
Rate	100	. Sinus tachycardia, rate 100	xis, rate >= 100
		. Borderline intraventricular conduction delayQRS	
		. Consider left atrial enlargement	
		. Anterolateral injury (ACUTE INFARCT)ST > .35 m	
	432		, , , ,

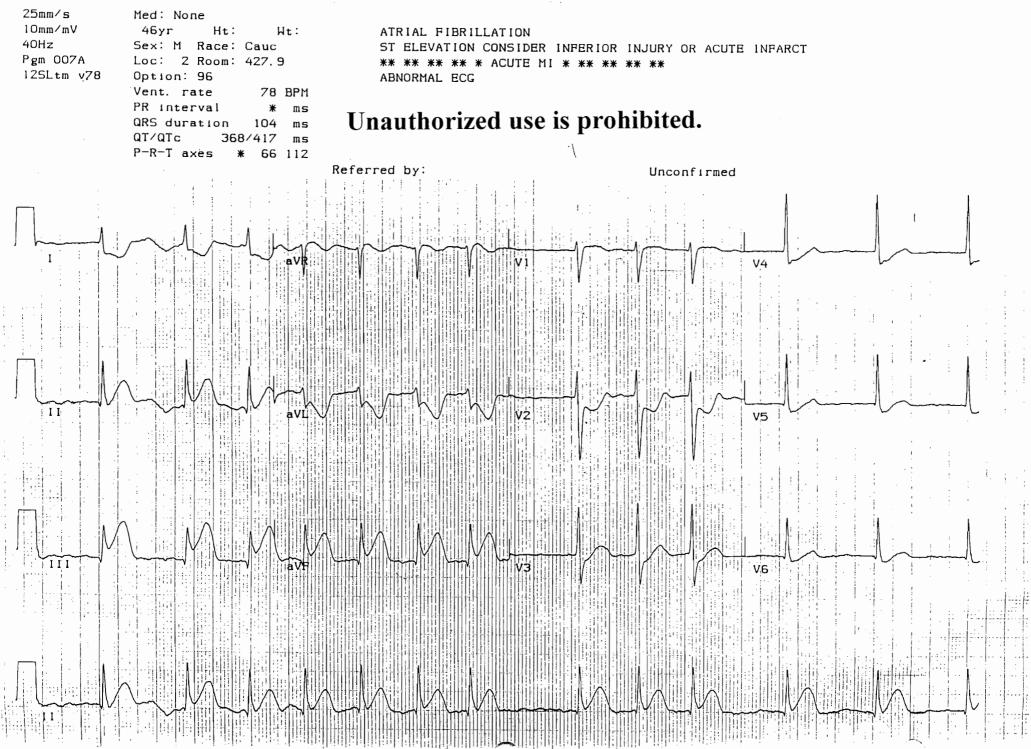
--AXIS--P 61

QRS

64

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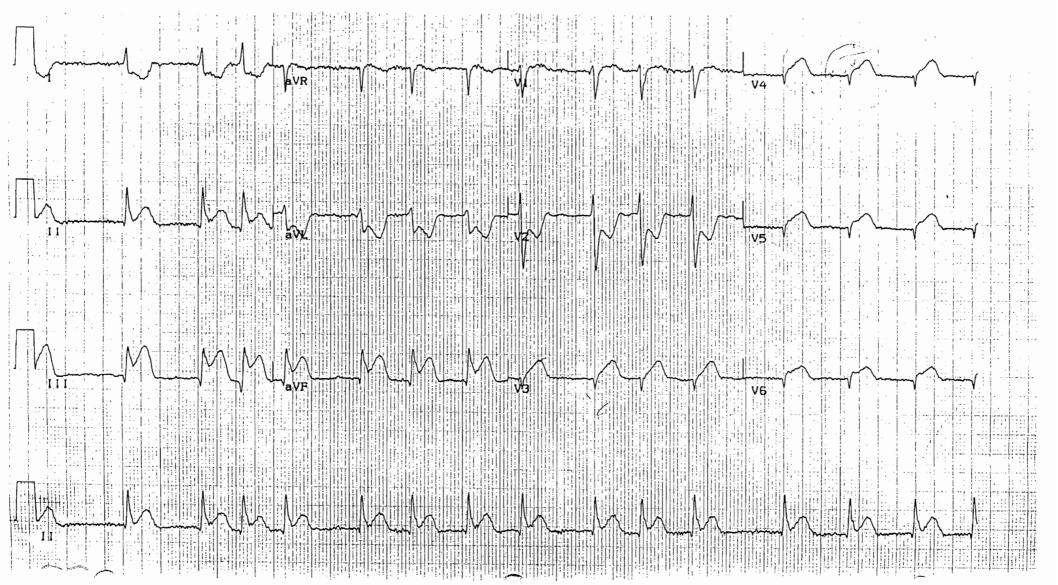




	ID:	15-MAR-99 04:49
25mm/s 10mm/mV 40Hz Pgm 107A 12SLtm v78	Med: Unknown 47yr Ht: Wt: Sex: M Race: Cauc Loc: 50 Room: 5009 Option: 2 Vent. rate 93 BPM PR interval % ms QRS duration 88 ms QT/QTc 328/404 ms P-R-T axes % 63 117	ATRIAL FIBRILLATION POSSIBLE ANTEROLATERAL INFARCT, AGE UNDETERMINED INFERIOR INJURY PATTERN ** ** ** ** ACUTE MI * ** ** ** ABNORMAL ECG Unauthorized use is prohibited.

Referred by:

Unconfirmed

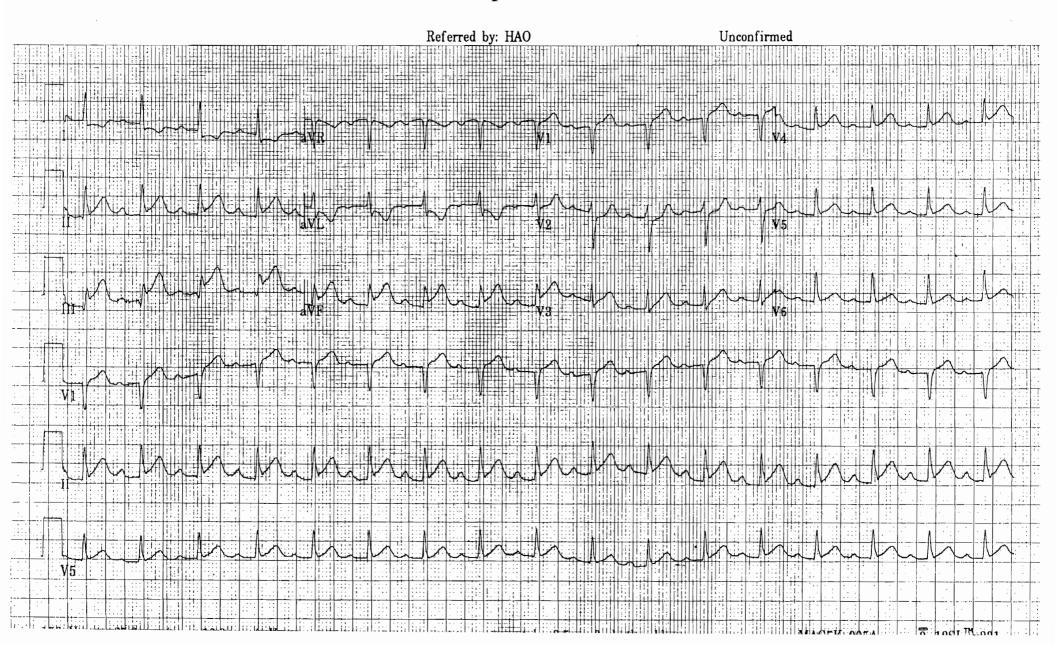


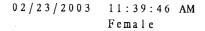
25-Mar-2003 17:19:56

26-Apr-1931Vent. rate101 bpm*** Age and gender specific ECG analysis ***FemalePR interval228 msSinus tachycardia with 1st degree AV blockQRS duration84 msST elevation consider inferior injury or acute infarctQT/QTc316/409 ms** ** ACUTE MI ** **P-R-T axes6650100

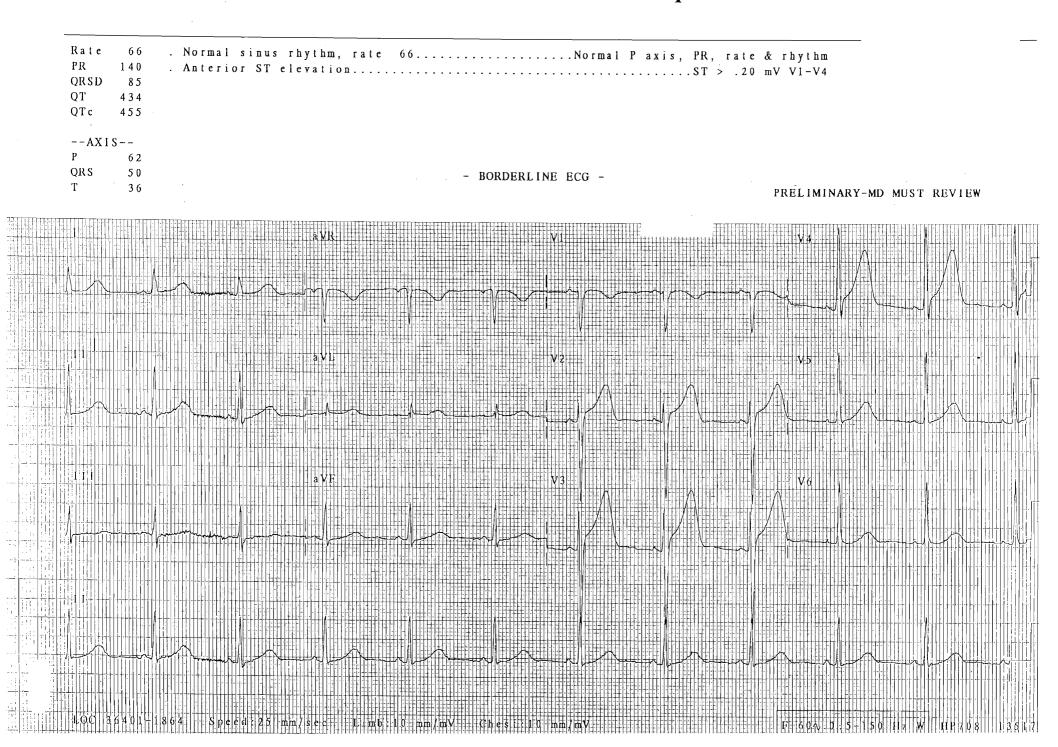
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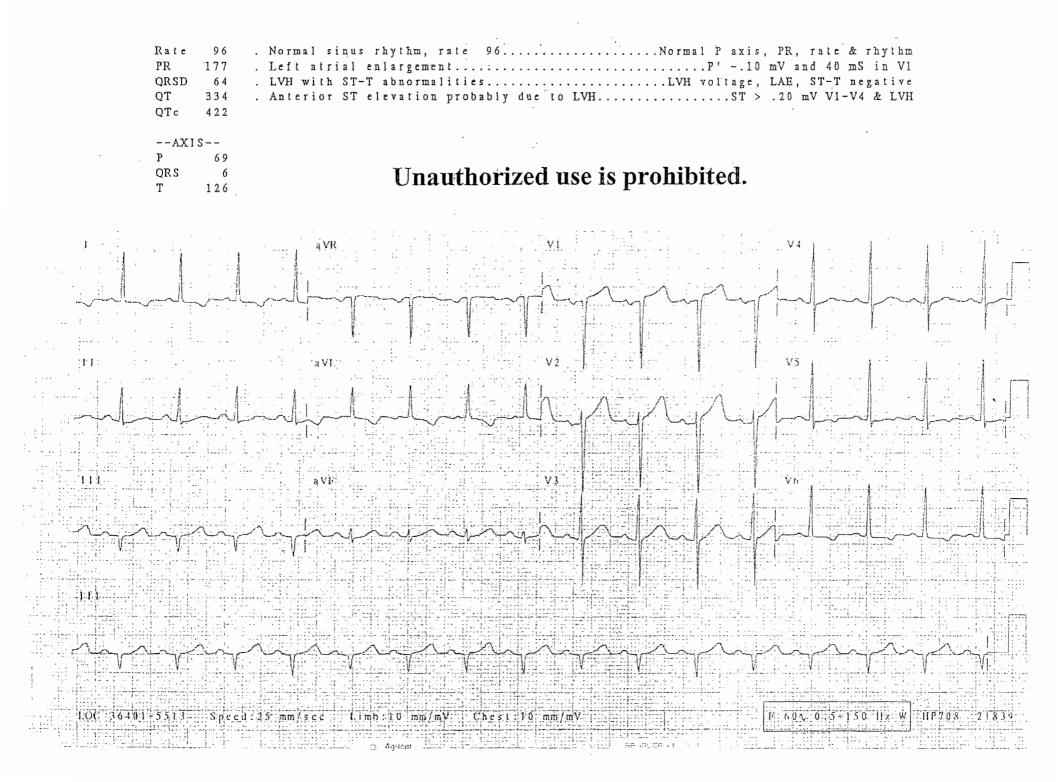
02/23/2003 01:41:55 PM Female

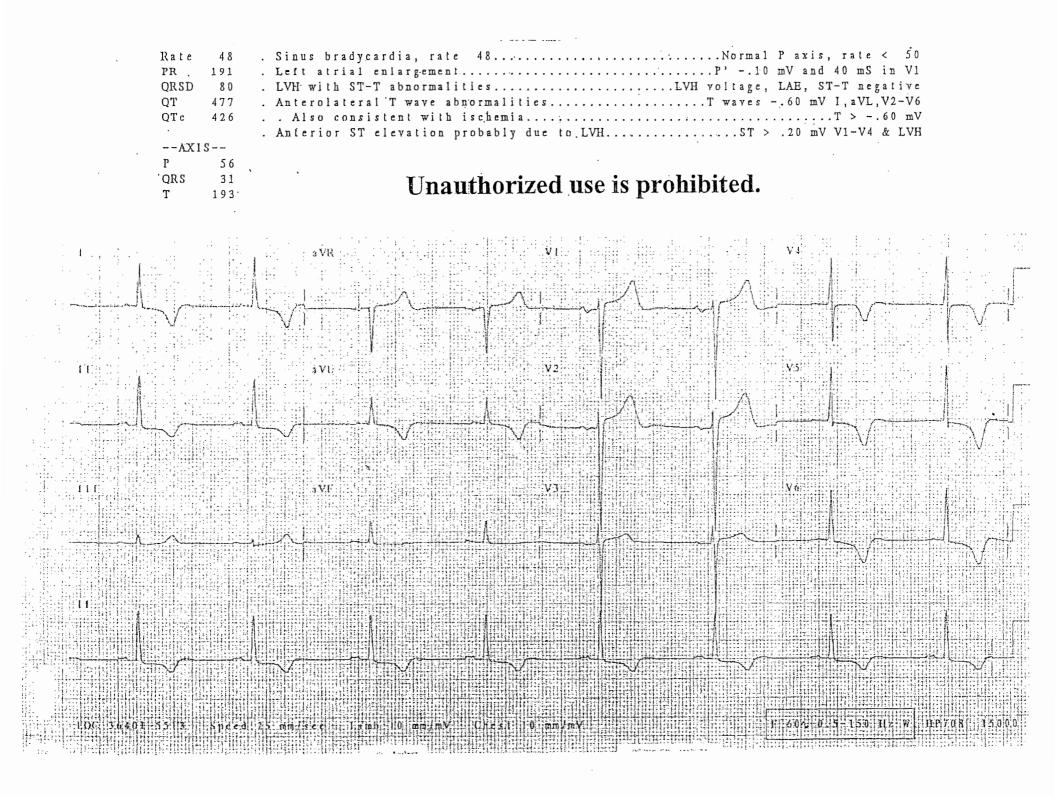
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Rate PR QRSE QT QTc	175 0 135 435	. Regular rhythm with unusual Paxis, rate 76Paxis not -30 to 120, norm. rate Left axis deviationQRS axis -31 to -90 Nonspecific intraventricular conduction delayQRS 120 mS or wider Consider right ventricular hypertrophyQ with large R in V1 or V2 Inferior infarct, possibly acuteQ's & ST>.10mV II,11I,aVF Acute Extensive Anterior infarctQ's in V1-V6, ST +.15 mV
Р	-83	
QRS	- 5 8	- ABNORMAL ECG -
Т	73	
		PRELIMINARY-MD MUST REVIEW
		\mathbf{v}_{1}



<u>111</u><u>v3</u><u>v6</u>

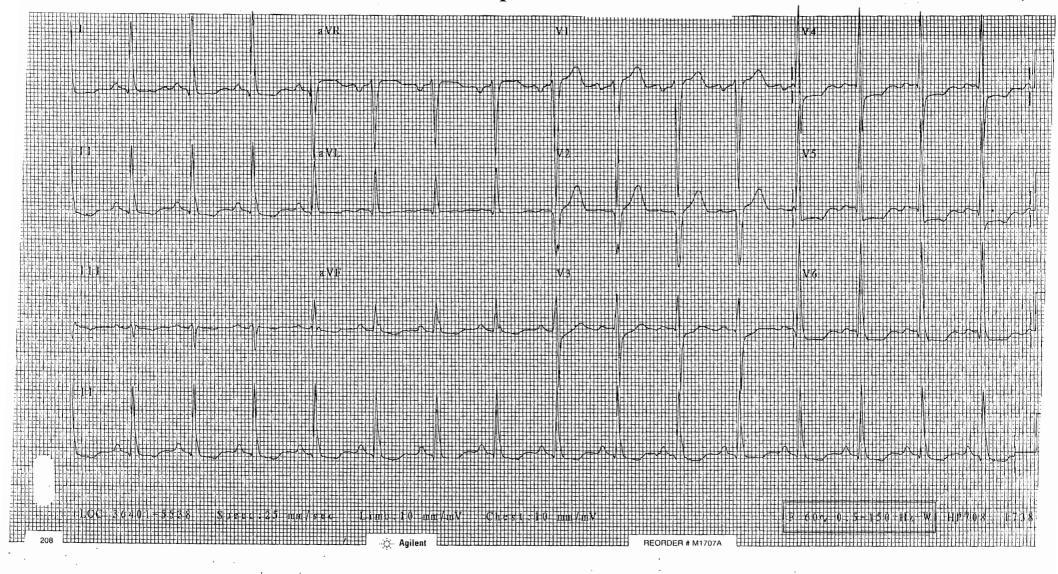




Rate	94	. Normal sinus rhythm, rate 94Normal Paxis, PR, rate & rhythm
PR	156	. Consider right atrial enlargementP > .24 mV limb lead
QRSD	106	. Consider left atrial enlargementPV110 mV or more negative
QT	338	. LVH with ST-T abnormalitiesLVH voltage, ST neg, QRS/VAT wide
QTc	423	. Anterior infarct, age indeterminateQ waves V2-V4, neg T's
		. Anterolateral ST depression,
AX)	(S	. Also consistent with subendocardial injury
P	47	
QRS	25	
Т	225	Unauthorized use is prohibited preliminar

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PRELIMINARY-MD MUST REVIEW



PR 16 QRSD 15 QT 46	0. Left bun 4 66	inus rhythm, rate dle branch block	70	Normal Paxis, QRS 120 mS, ter	PR, rate & rhythm minal forces left		
QTc 50		Unauth	orized use is	prohibited.		Requested by	′:
AXIS P QRS -1 T 15	2 8		- ABNORMA	L ECG -	PRELIMI	NARY-MD MUST REV	/IEW
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13:15:00

	(71 уг)	Vent. rate	78	BPM	Normal sinus rhythm
Female	Black	PR interval	146	ms	Left atrial enlargement
		QRS duration	179	ms	Non-specific intra-ventricular conduction block
		QT/QTc	463/527	ms	Possible Lateral infarct, age undetermined
Loc:7		P-R-T axes	39 -50	104	Inferior infarct, age undetermined
					Abnormal electrocardiogram

Unauthorized use is prohibited.

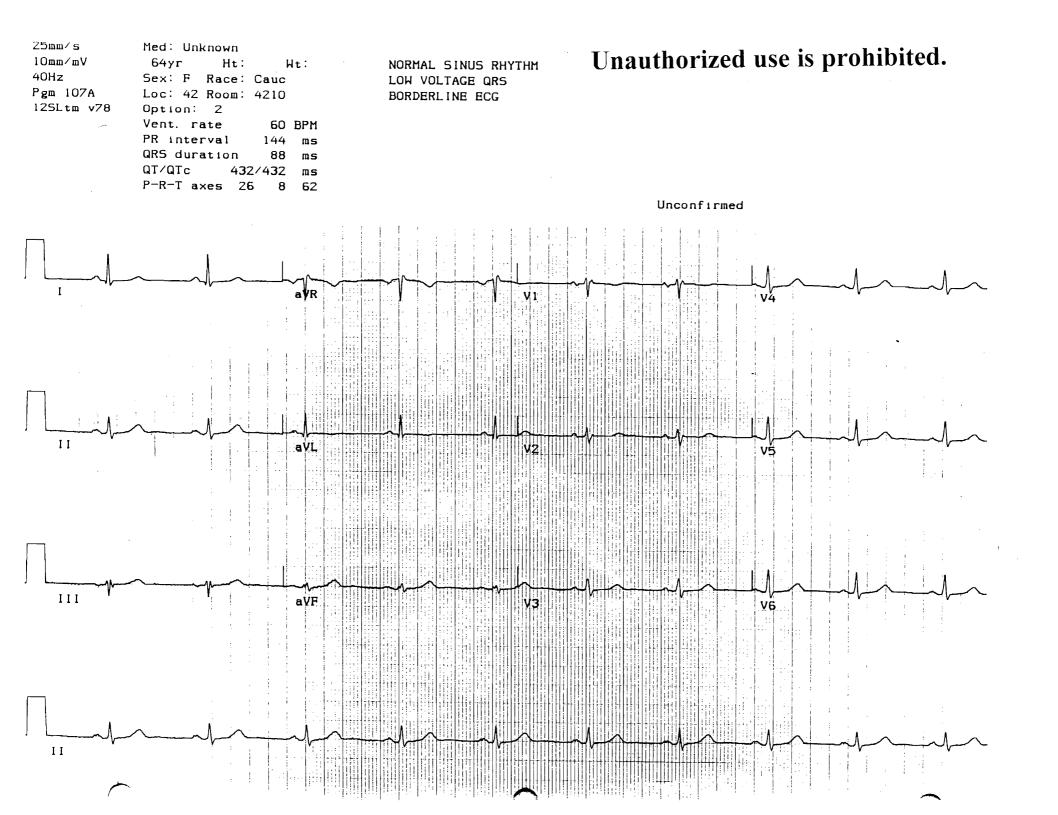
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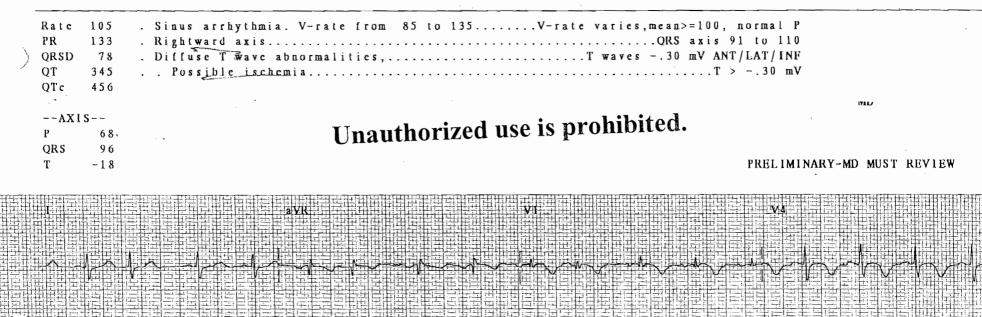
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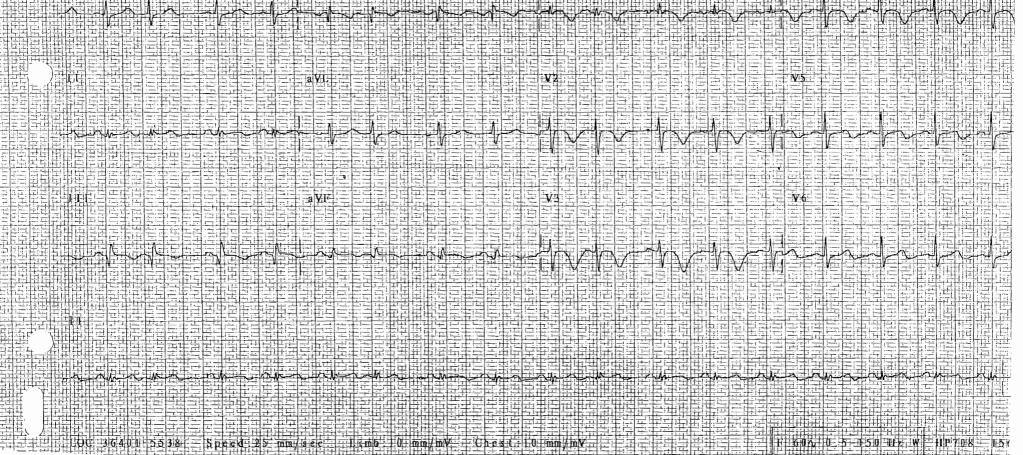
25mm/s 10mm/mV 100Hz 005E 12SL 86 CID: 1

SID: 107 EID:42 EDT: 20:20 16-AUG-2006 ORDER:

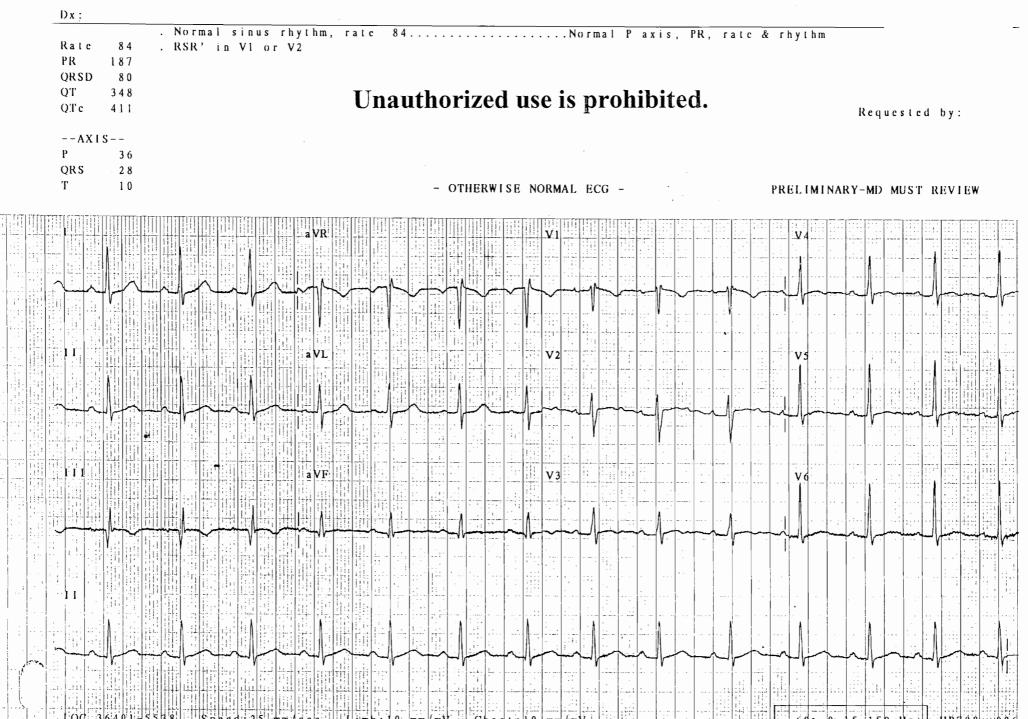
Rate 74 PR 174 QRSD 112 QT 355	Normal sinus rhythm, rate 74	ate & rhythm :40 inferior in V5 or V6
QTc 394		Requested by:
AXIS P 46 QRS -60 T 48	Unauthorized use is prohibited.	PRELIMINARY-MD MUST REVIEW
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		No.
	X (83) Sizes 225 and see Lines 30 and any Chester 10 and any	7







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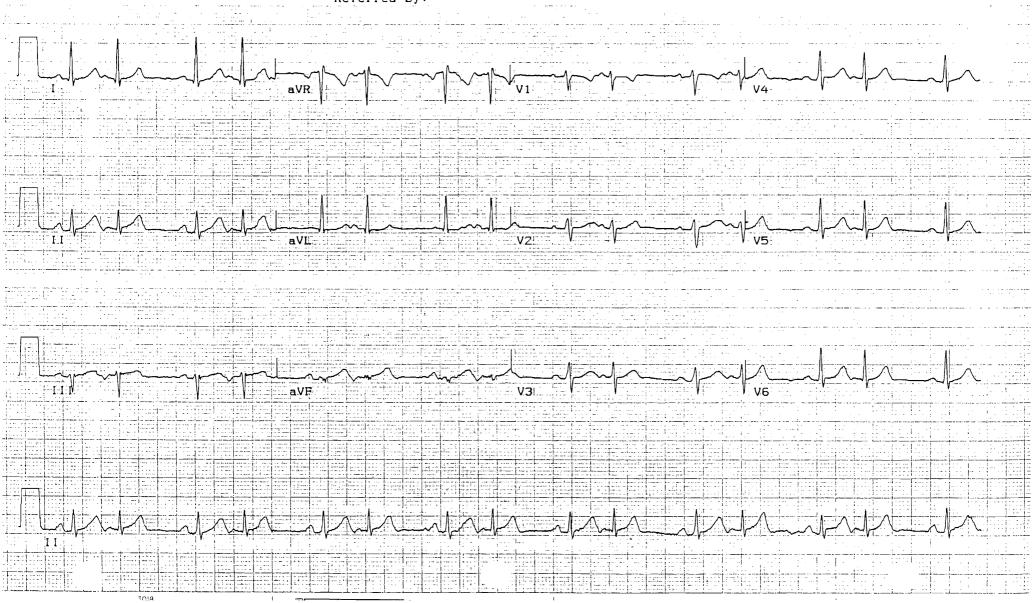


25mm/s	Med:		·	
10mm∕mV	34yr	Ht:	н	t:
40Hz	Sex: F Ra	ce:	Cauc	
Pgm 007A	Loc: 2 Ro	om:	427.9	
12SLtm v78	Option: 84			
	Vent. rate		90	BPM
	PR interva	1	152	ms
	QRS durati	оп	80	ms
	QT/QTc	3 72	/452	ms
	P-R-T axes	58	-2	46

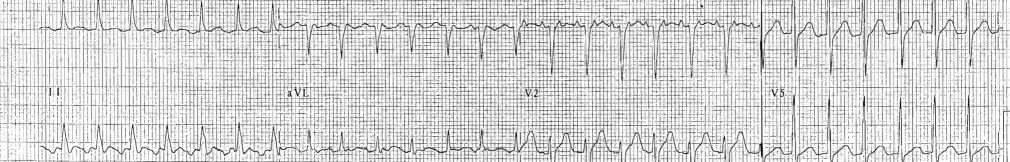
NORMAL SINUS RHYTHM WITH FREQUENT PREMATURE ECTOPIC COMPLEXES OTHERWISE NORMAL ECG

Unauthorized use is prohibited.

Referred by:



Rate	164	. Supraventricular tachycardia, rate = 164V-rate > (220-age) or 150
PR	124	. Nonspecific Inferior T wave abnormalities
QRSD	98	Cannot exclude ischemiaT >20 mV
QT	260	
QTc	429	
AX I	S – –	Unauthorized use is prohibited.
Р	84	
QRS	28	- ABNORMAL ECG -
Т	- 5 4	PRELIMINARY-MD MUST REVIEW
		$- \frac{2\sqrt{n}}{2}$



1.1.1

LOC 36401-1864 Speed: 25 mm/sec Limb: 10 mm/mV

F 605 0-5-150 Hz W HP708 1443

V6-

Rate PR QRSD QT QTc AX P	1 2 1 2 8 1 4 4 8	. Atrial fib. w/ . Vertical axis, . Right bundle b	unusual for ranch block.	age	QRS 12	QRS axis 81 20 mS, term	to 90 & age > inal forces ri	40	
Q R S T .	8 8 2 9	· ·	د .	- AB)	NORMAL ECG -		PREI	IMINARY-MD MUST RE	VIEW

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