COMMON ARRYTHMIAS

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CARDIAC SCIENCES PROGRAM
PROGRAMME DES SCIENCES CARDIAQUES

UNIVERSITY OF MANITOBA
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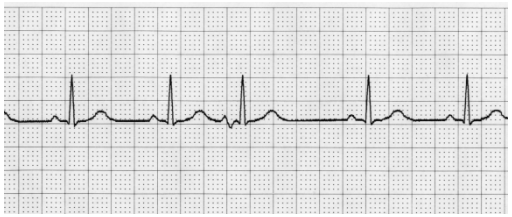
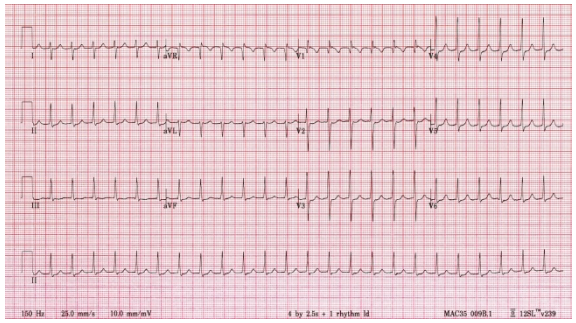
DISCLOSURES

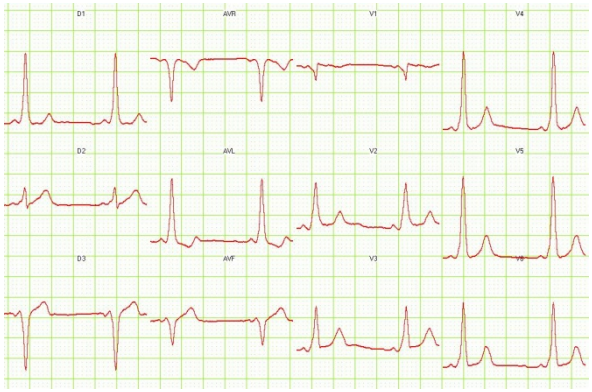
- There is no conflict of interest and there is nothing to disclose



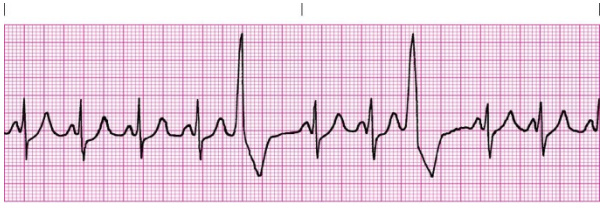
Supraventricular premature beats



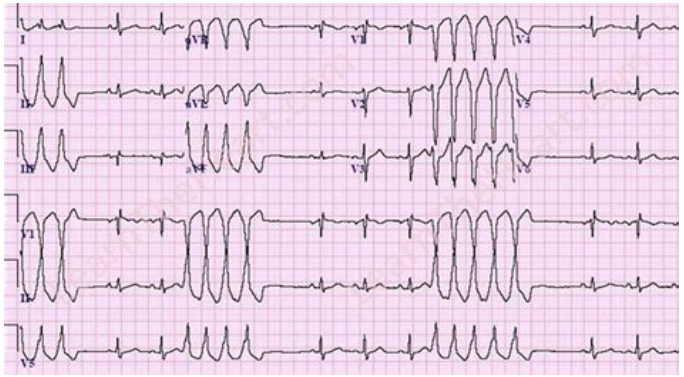
Supraventricular tachycardia



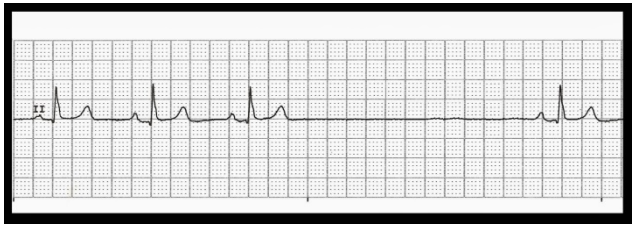
Wolff-parkinson-white syndrome (WPW)



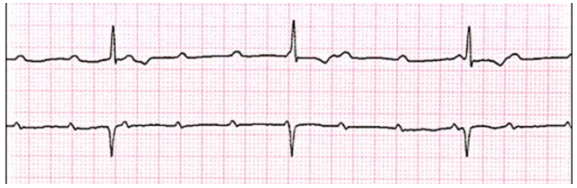
PVCs



Non-sustained Ventricular tachycardia



Sinus pauses



Heart blocks (AV nodal Blocks)

My patient has Premature Ventricular Complexes (PVCs)

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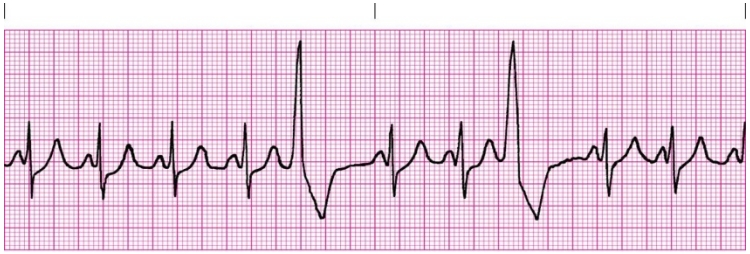
What to do next?



- PVCs are common and seen in a broad spectrum of the population.
- These are more commonly seen in patients with structural heart disease
- But it is not uncommon to see them in patients without structural heart disease.

High Risk PVCs

- Multifocal PVCs
- Two consecutive PVCs (couplets)
- Three or more consecutive PVCs (salvos/non-sustained ventricular tachycardia)
- "R-on-T" phenomenon

- Over last few decades, it has become evident that prognosis relates only to the underlying structural heart disease and the degree of left ventricular dysfunction, not to the complexity of the PVCs.
- These complex PVCs may be a marker of presence of underlying cardiac disease

Prevalence of PVCs in General population

*Most Frequent Major Abnormalities—Rates/1000**

Diagnosis	16-19	20-24	25-29	30-34	35-39	40-44	45-49	50 & over	Total	Rate per 1,000
Atrial rhythm	14.5 (103)	8.9 (332)	4.6 (101)	2.6 (34)	2.0 (45)	2.9 (54)	0.9 (2)	1.6 (1)	672	5.5
Atrial premature contractions	6.2 (44)	4.8 (181)	4.0 (87)	3.1 (41)	3.9 (86)	4.2 (78)	4.8 (10)	11.5 (7)	534	4.3
Nodal premature contractions	1.6 (12)	2.4 (90)	2.1 (53)	1.2 (16)	1.9 (42)	2.5 (47)	0.4 (1)	2.7 (2)	263	2.1
Ventricular premature contractions	4.6 (33)	6.2 (232)	5.7 (124)	8.3 (108)	8.2 (182)	11.8 (218)	19.0 (39)	21.7 (16)	952	7.8
First-degree AV block	6.0 (43)	7.4 (275)	6.2 (136)	5.0 (65)	5.9 (132)	7.1 (131)	4.8 (10)	13.6 (10)	802	6.5
Wolff-Parkinson-White	1.8 (13)	1.1 (54)	1.9 (42)	2.2 (29)	1.3 (30)	0.9 (17)	0.9 (2)	0 (0)	187	1.5
Right bundle-branch block	0.5 (4)	1.2 (48)	1.3 (29)	2.0 (27)	2.7 (60)	2.9 (54)	1.9 (4)	0.9 (7)	231	1.8
Intraventricular conduction defect	1.9 (14)	4.2 (159)	4.0 (87)	4.2 (55)	4.8 (107)	3.7 (68)	4.8 (10)	6.8 (5)	505	4.1
Ventricular fusion beats	0.4 (3)	0.2 (10)	0.5 (12)	0.6 (8)	0.4 (10)	0.5 (10)	0 (0)	2.7 (2)	55	0.7
Nonspecific T waves	15.1 (107)	9.9 (368)	10.4 (224)	8.9 (114)	12.1 (267)	14.4 (266)	18.1 (37)	29.9 (22)	1405	11.5
AV dissociation with AV nodal rhythm	3.1 (22)	1.3 (49)	0.37 (8)	0 (0)	0.09 (2)	0.17 (3)	0.5 (1)	0 (0)	85	0.7

Electrocardiographic Findings in 122,043 Individuals
By ROLAND G. AND LAWRENCE E. Circulation 1962; 25:947.

The prevalence of PVC on a 2 min EKG on middle age adults (45 -65 yrs of age) was approximately 6%

Table I. Age-adjusted prevalence (percent and number) of PVCs on 2-minute ECG, by PVC frequency and morphologic characteristics, ARIC cohort baseline examination, 1987-1989

	Men		Women	
	African American (n = 1542)	**White (n = 5220)**	**African American (n = 2462)**	**White (n = 5846)**
Any PVC	8.2 (127)	6.7 (379)	6.9 (159)	4.7 (275)
Single	3.2 (45)	2.9 (149)	2.9 (63)	1.9 (109)
Frequent	4.4 (65)	3.1 (172)	3.1 (71)	2.5 (141)
Complex	0.9 (17)	1.0 (58)	0.9 (25)	0.5 (25)
Frequent/complex	5.3 (82)	4.0 (230)	4.2 (96)	2.9 (166)

Prevalence is adjusted to age 55 years; single = only one PVC, without R on T PVCs on 2-minute ECG; frequent = 2 or more PVCs, without complex forms; complex = consecutive, multiform or R on T PVCs.

Prevalence of premature ventricular contractions in a population of African American and white men and women: The Atherosclerosis Risk in Communities (ARIC) Study Ross J., Wayne E. et al. Am Heart J 2002;143:535-40.

Prevalence of PVCs on ambulatory monitoring

Table 4—*Ambulatory Monitoring in Normal Subjects*

Author	Year	Age	N	%VA	%SVA	% Complex Arrhythmia*
Hinkle et al[8]	1969**	55	283	62	76	37
Raftery et al[9]	1976	20-79	53	17	23	few
Clarke et al[10]	1976	16-65	86	73	5†	12
Brodsky et al[7]	1977	23-27	50	50	56	24
Present study	1978	60-84	13	100	100	77

*Multiform, couplets, salvoes, R on T, bigeminy, frequent
**Six hour "stress" monitoring
†Supraventricular tachycardia not listed
N = number
%VA = percentage with ventricular arrhythmias
%SVA = percentage with supraventricular arrhythmias

- The prevalence of PVCs is related
  - to the study population,
  - the method used to investigate and the
  - duration of monitoring/test.
- In patients with no known apparent heart disease, PVCs occur in < 1% on a routine 12-lead ECG
- In 24-hour ambulatory monitoring, >50% of apparently healthy people have occasional PVCs and upto 1-6% had >50 PVC/24 hours
- There is an age-related increase in the prevalence of PVCs in normal individuals and those with underlying heart disease
- The occurrence of frequent PVCs accounting for more than 20 percent of overall heart beats is rare

Conditions associated with more frequent PVCs.

CARDIAC CONDITIONS

- Acute myocardial infarction
- Heart failure/cardiomyopathy
- Myocarditis
- Hypertrophic cardiomyopathy
- Congenital heart disease
- Hypertension with left ventricular hypertrophy

- High grade PVCs are more commonly seen in advanced form of underlying cardiac disease, which has worse prognosis.

- This suggest that in this patient population, PVCs are just the marker of more advanced disease and it is the severity of the disease which is related to the prognosis and worse outcomes

Conditions associated with more frequent PVCs.

NON-CARDIAC CONDITIONS

- Chronic obstructive pulmonary disease
- Sleep apnea syndromes
- Pulmonary hypertension
- Other pulmonary diseases and
- endocrinopathies (thyroid, adrenal or gonadal abnormalities).

Triggers to provoke PVCs

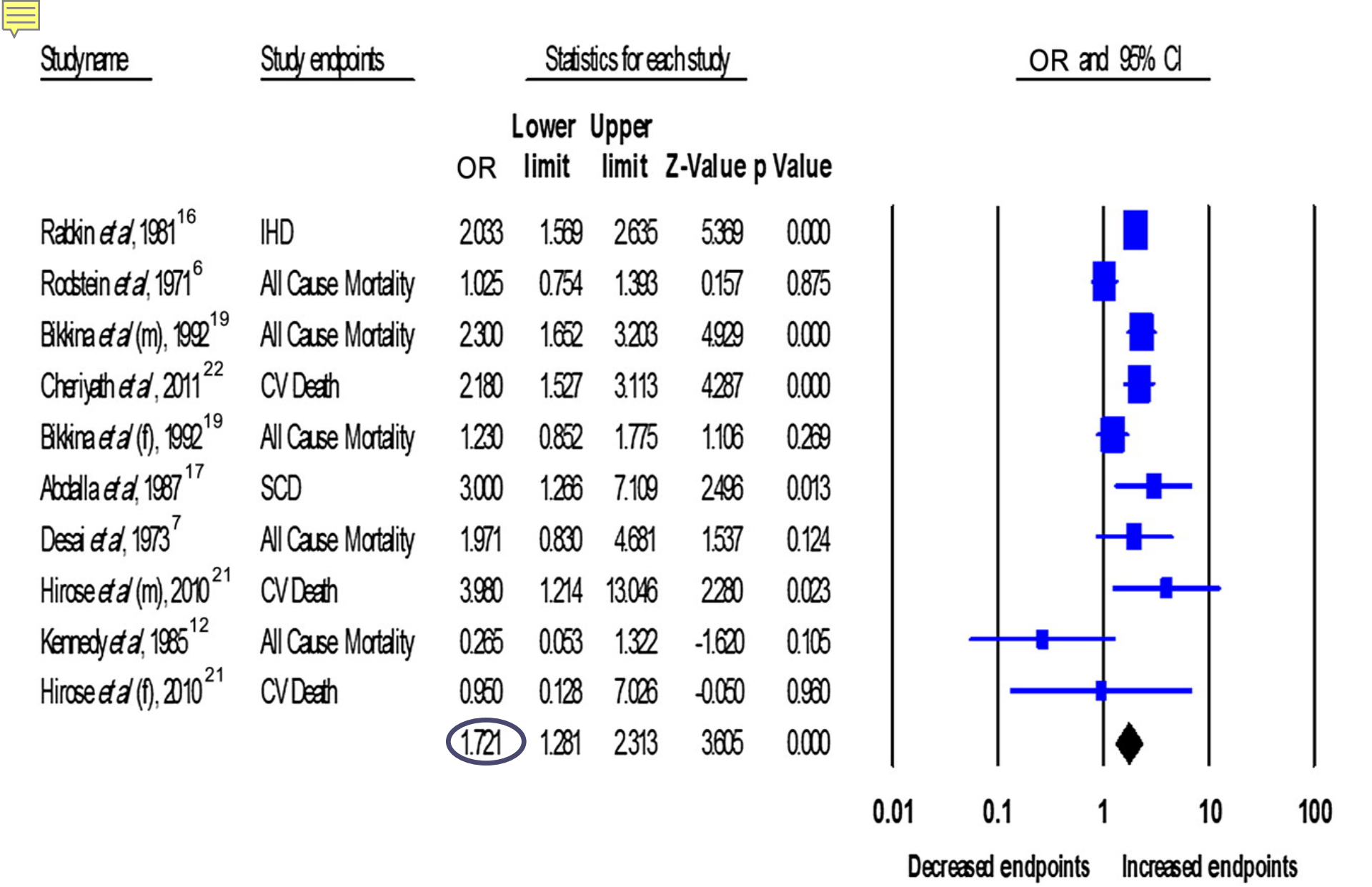
- Alcohol
- Tobacco
- Caffeine
- Anemia
- Anxiety
- Exercise
- High blood pressure
- Stimulants, such as sympathomimetic agents (eg, beta-agonists, decongestants, antihistamines)
- Illicit drugs (eg, cocaine, amphetamines)

Symptoms

- Most patients are asymptomatic
- The most common symptom is palpitations
- Rarely, PVCs can result in hemodynamic compromise, causing symptoms of dizziness, presyncope and decrease in exercise capacity
  - When they occur frequently in a patient with severely depressed left ventricular function or
  - When they are associated with an underlying bradycardia
  - When they are extremely frequent

- Extremely frequent PVCs (typically > 15-20% of all beats) can result in a reversible cardiomyopathy, even in the absence of sustained ventricular arrhythmias or symptoms
- This effect is most pronounced in younger patients without comorbidities, suggesting that PVCs may be an important cause of "idiopathic" HF.
- Elimination of PVCs using catheter ablation or medications often leads to normalization of cardiac function

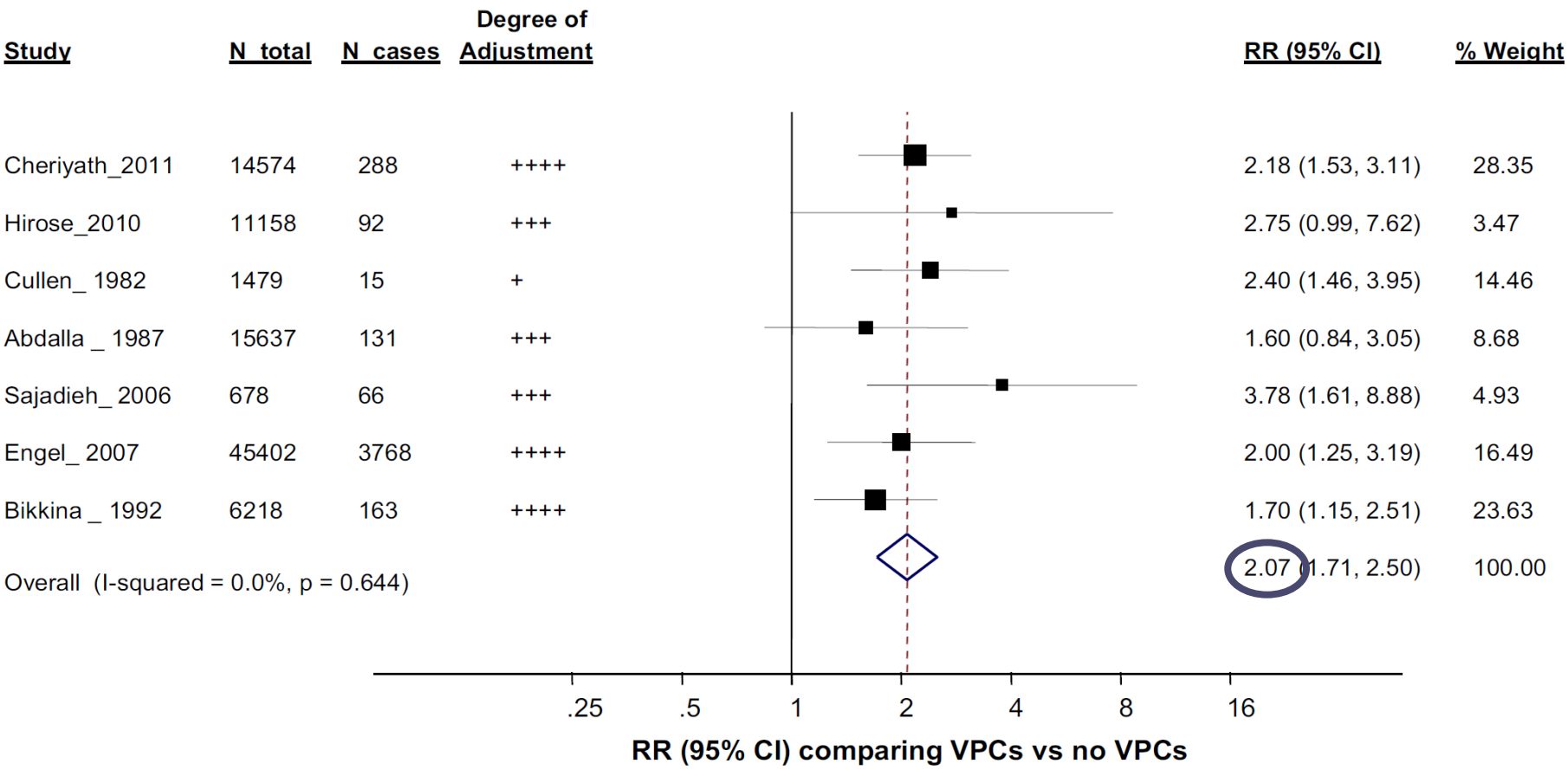
PROGNOSIS



The prognostic significance of premature ventricular complexes in adults without clinically apparent heart disease: a meta-analysis and systematic review Lee V, Hemingway H. et al. Heart 2012;98:1290e1298

Association of Frequent PVCs with risk for SCD

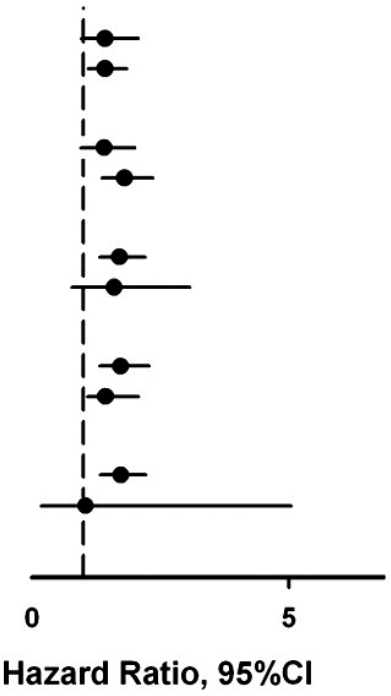
Frequent PVCs were defined as occurring ≥ 1 time during a standard electrocardiographic recording or ≥ 30 times over a 1-hour recording.



Meta-Analysis of Ventricular Premature Complexes and Their Relation to Cardiac Mortality in General Populations Ataklte F, Erqou S, MD et al. Am J Cardiol 2013;112:1263e1270

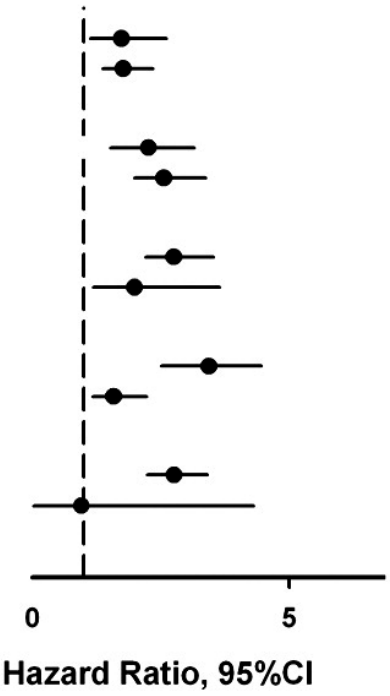
Monomorphic PVC & Mortality

Variable	Hazard Ratio (95%CI)	P value
Age		
≤ 65	1.42(0.96-2.10)	0.080
> 65	1.41(1.07-1.85)	0.013
Gender		
Female	1.40(0.97-2.03)	0.077
Male	1.88(1.42-2.48)	<0.001
DM		
No	1.75(1.38-2.22)	<0.001
Yes	1.63(0.88-3.03)	0.123
HTN		
No	1.77(1.32-2.36)	<0.001
Yes	1.45(1.03-2.06)	0.035
CKD		
No	1.74(1.39-2.17)	<0.001
Yes	1.08(0.23-5.02)	0.919



Polymorphic PVC & Mortality

Variable	Hazard Ratio (95%CI)	P value
Age		
≤ 65	1.75(1.17-2.62)	0.006
> 65	1.83(1.43-2.36)	<0.001
Gender		
Female	2.20(1.54-3.15)	<0.001
Male	2.61(2.01-3.38)	<0.001
DM		
No	2.78(2.23-3.47)	<0.001
Yes	2.03(1.14-3.64)	0.017
HTN		
No	3.34(2.56-4.34)	<0.001
Yes	1.63(1.17-2.26)	0.004
CKD		
No	2.76(2.25-3.40)	<0.001
Yes	0.89(0.19-4.18)	0.878



Long-term outcome of multiform premature ventricular complexes in structurally normal heart
Chin-Yu Lin, Shih-Lin Chang, et al. International Journal of Cardiology 180 (2015) 80–85

PROGNOSIS

- Though, traditionally, PVCs are often thought to be of minimal clinical significance, particularly in patients without a history of cardiac disease, most studies have reported an increased mortality in patients with PVCs, especially with frequent and complex PVCs in general population.

- But, these studies, though, were done in adults without clinically apparent heart disease, but most studies did not use advanced tests to rule out structural heart disease.

The prognostic significance of premature ventricular complexes in adults without clinically apparent heart disease: a meta-analysis and systematic review Lee V, Hemingway H. et al. Heart 2012;98:1290e1298

PROGNOSIS

- The 5 uncontrolled studies which studied patients with lower cardiovascular risk and has a high usage of echocardiography and stress test, observe zero mortality, SCD, VT or syncope events in their PVC cohort over an average of 6.9 years.

- This may suggest that the poor prognosis may be secondary to inadvertent inclusion of patients with occult structural heart disease, the population in which PVCs are known to confer adverse outcomes

The prognostic significance of premature ventricular complexes in adults without clinically apparent heart disease: a meta-analysis and systematic review Lee V, Hemingway H. et al. Heart 2012;98:1290e1298

PROGNOSIS

- Also, prophylactic treatment of asymptomatic PVCs or non-sustained VT, in patients without any structural heart disease, has not been shown to reduce mortality.
- This holds true for most of the cases with underlying structural heart disease, except for few exceptions
- As such, the presence of significant PVCs should alert the clinician to potential coexistent cardiac disease, which may require additional clinical assessment or therapy.

In patients with ***frequent and unexplained PVCs***, the following evaluation should be performed:

- **Thorough history and physical exam**
  - To assess for any underlying structural heart disease (HTN, Ischemic heart disease, CHF, valve disease)
  - With documented nocturnal PVCs, patient should be assessed for sleep apnea
  - History of use of triggers like alcohol or caffeine-containing beverages, or illicit drugs, or use of other stimulants etc
  - For family history of sudden cardiac death and cardiomyopathy
- **Laboratory testing**
  - Electrolyte levels,
  - Thyroid stimulating hormone [TSH]

- **EKG**
  - Any evidence of Previous MI
  - Conduction abnormalities
  - LVH
  - Significant repolarization changes

- **24-hour ambulatory (Holter) monitor**
  - To quantify the frequency of PVCs
  - Any sustained or non-sustained VT
  - To determine the morphology of PVCs (if they are monomorphic or polymorphic)

- **Echocardiography**
  - If history suggestive of cardiac disease or there are frequent or complex PVCs, echo should be performed to assess cardiac structure and function
- **Exercise treadmill stress test**
  - To evaluate the response of the PVCs to exercise
  - To determine if sustained or non-sustained ventricular tachycardia (VT) can be induced with exercise
  - To screen for underlying ischemic heart disease

TREATMENT

- In persons found to have frequent PVCs, further management is based on the
  - presence or absence of underlying structural heart disease

    and/or

  - symptoms

In absence of structural heart disease

- **In Patients with low grade ectopy (including bigeminy)**
  - **Avoiding exposure** to possible triggers should be advised, although this strategy has not been a consistently effective
  - **Reassurance** regarding the benign nature of ventricular ectopy
  - **Regular follow-up by family MD**
  - If still symptomatic, then treatment with **a beta-blocker and/or a calcium channel blocker** should be initiated

- **Beta blockers** tends to suppress PVCs due to excess sympathetic stimulation or increased catecholamines
- In other conditions, it may not reduce frequency, but may reduce symptoms.
- There is no clear evidence that suppression of PVCs with beta blockers or antiarrhythmic drugs improves overall survival in patients who have no symptoms and have not had a major arrhythmic event.
- The lowest dose of beta blockers that relieves symptoms should be used in order to minimize side effects, with the exception of patients with a prior MI or LV dysfunction

In absence of structural heart disease

- **Consider Cardiology referral**
  - If still symptomatic after treatment and reassurance
  - Unable to tolerate beta-blocker and/or a calcium channel blocker
  - If frequent and complex ventricular ectopy
  - Patient with high burden of PVCs (generally >15 percent of beats or >10,000 VPBs/24 hours) should also be **referred earlier to cardiology** because of risk of developing cardiomyopathy

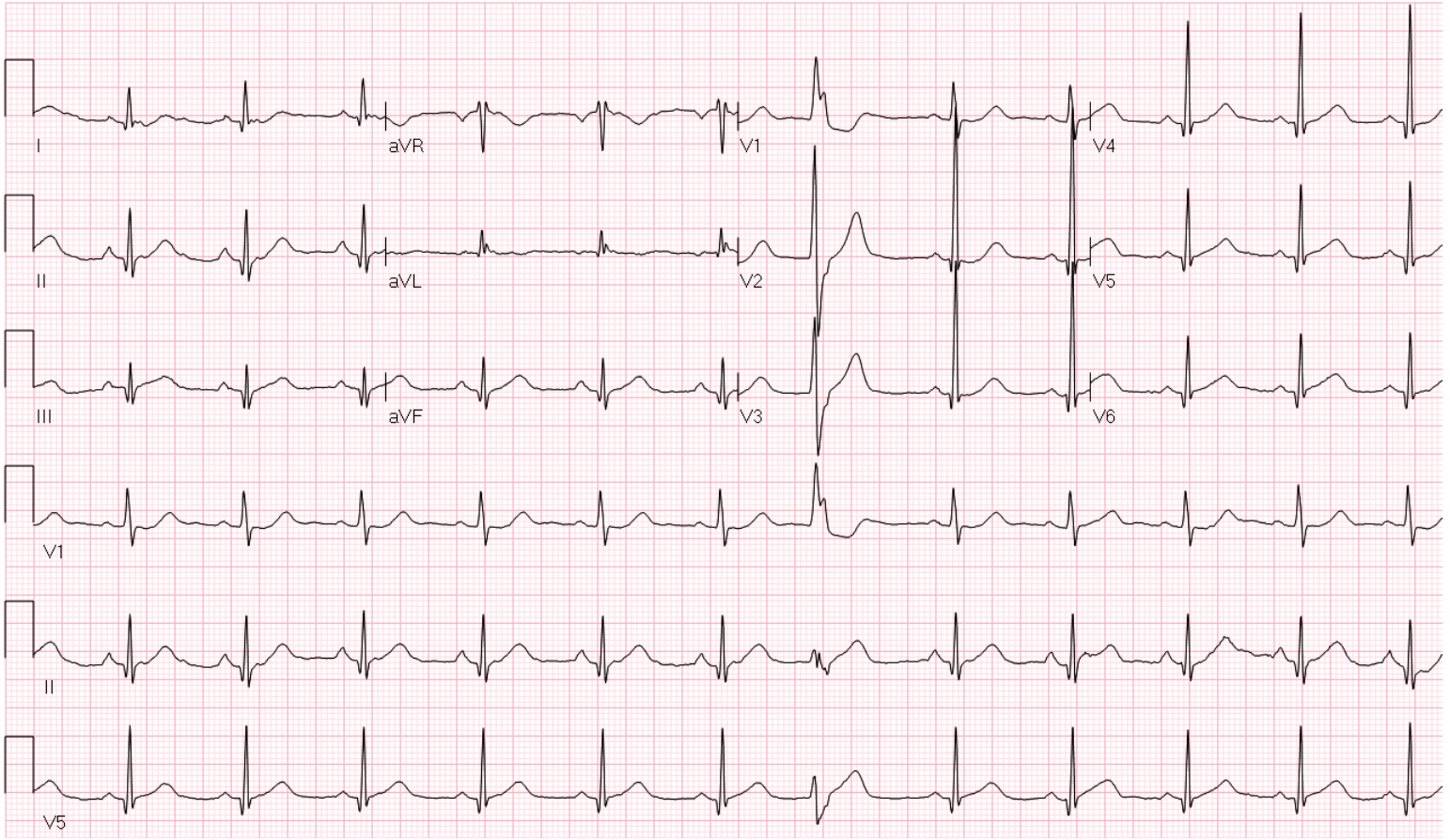
In patients with pre-existing/or newly diagnosed cardiac disease

- Patient should be **adequately treated for his underlying cardiac disease** (eg. beta blockers, ACEi, diuretics for CHF, good control of BP with hypertension)
- What is the **cardiac function** (LVEF > 50%, LVEF < 40%
- Consider treatment with **beta blockers**
- Consider **cardiology referral** for complete assessment of cardiac status and to be assessed for possible treatment with antiarrhythmic therapy and catheter ablation

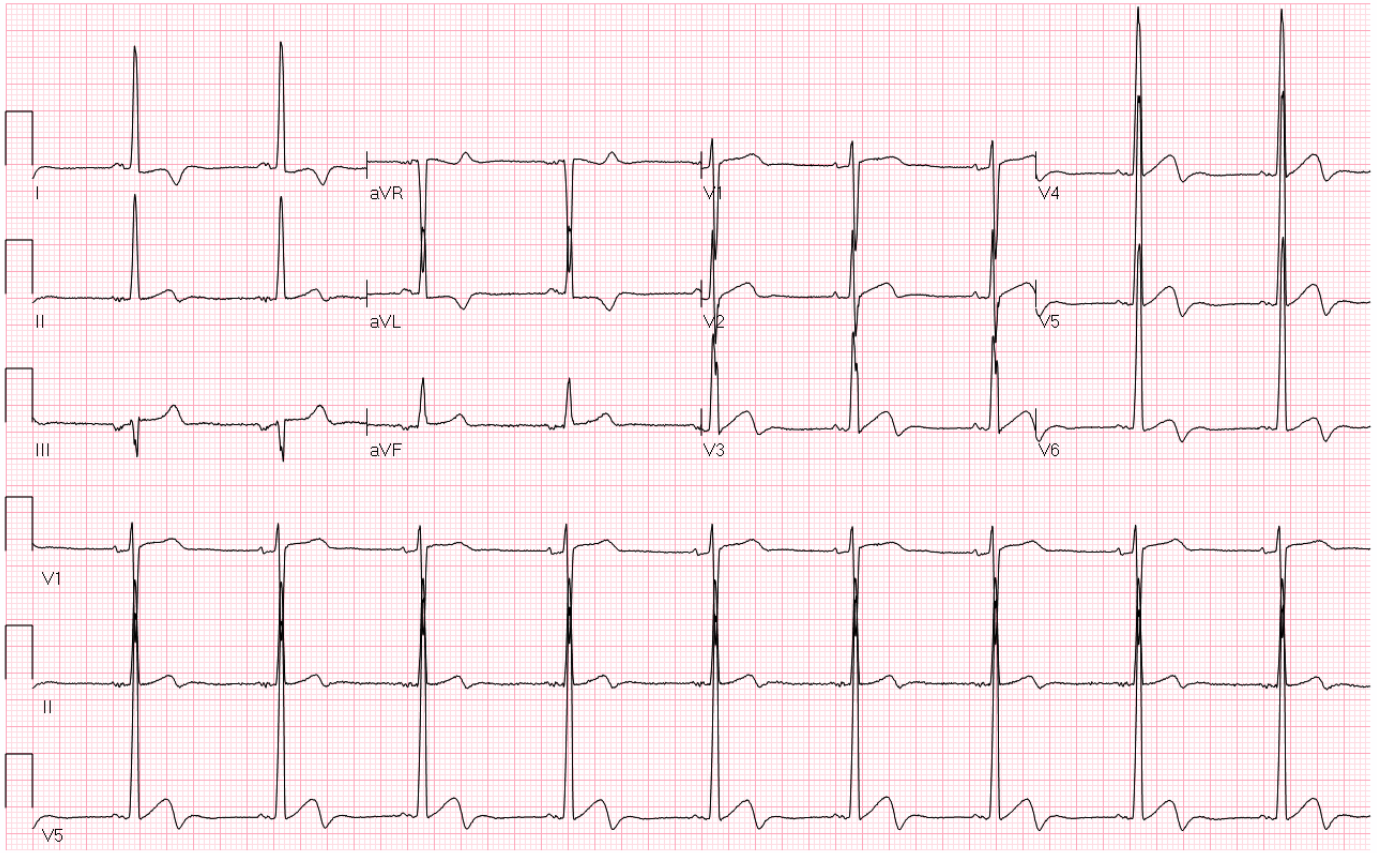
CONCLUSION

- PVCs are one of the most common arrythmia
- Current belief is, that presence of PVCs in itself does not affect survival.
- Complex and frequent PVCs may be a marker of underlying disease and it is the underlying disease which affects survival
- Treating PVCs with any of the drugs have not shown to reduce mortality
- **Thus, PVCs should be evaluated in context of company it keeps**
  - ***In absence of any structural heart disease***, the only indication to treat will be symptoms control, otherwise reassurance and routine clinical follow-up
  - ***In presence of heart disease***, the most important management is adequate treatment of underlying disease

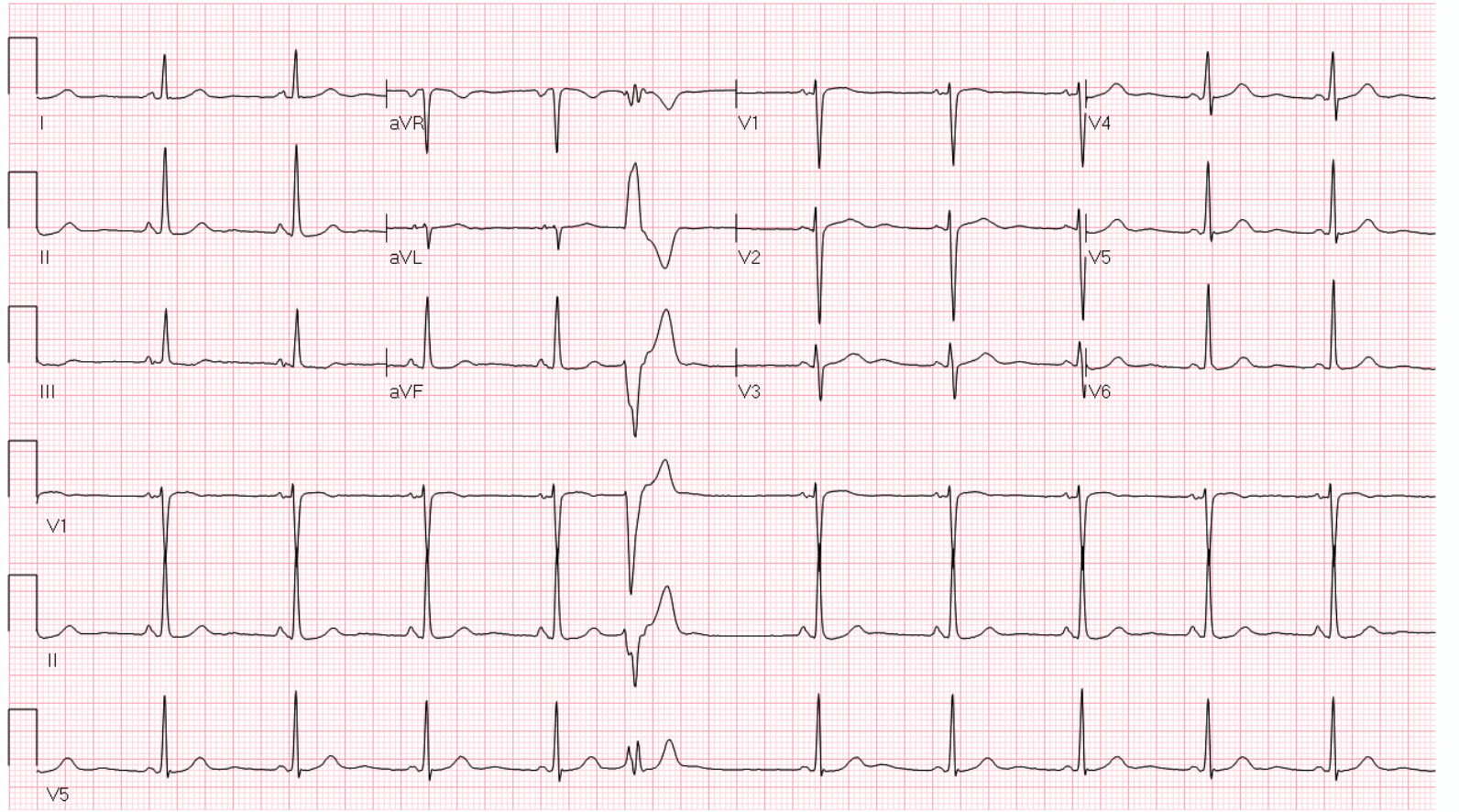
- 50 yr old
- Hx of HTN, DM and Hx of MI 4 years ago
- Feels intermittently light headed



- 37 yr old
- No significant PMH
- Family Hx of sudden cardiac death (Father died suddenly at age of 43)
- Complaining of palpitation



- 58yr old
- No PMH
- Healthy active gentleman
- Routine EKG



Thank you